Medical Library

Number, 12/6 net. [No. 47.

commence in JULY. Title and Index
for Binding, 4/- net; or the Volume
four numbers, for 7/6 net, post free.

British Journal of Surgery

UNDER THE DIRECTION OF THE FOLLOWING
EDITORIAL COMMITTEE:

Sir BERKELEY G. A. MOYNIHAN, Bart., K.C.M.G., C.B. (Leeds), *Chairman.*

[illegible] E. ARMSTRONG, C.M.G. (Liverpool)
[illegible] HAMILTON A. BALLANCE, K.B.E., C.B. (Norwich)
[illegible] GILBERT BARLING, Bart., K.C.B., C.B.E. (Birmingham)
[illegible]AMES BERRY (London)
[illegible]ARTHUR H. BURGESS (Manchester)
[illegible]. FOWELL CONNOR, R.A.M.C., L.R.C.P., I.M.S. (Calcutta)
[illegible]OSEPH P. DOBSON (Leeds)
[illegible] THOS. CRISP ENGLISH, F.C.S. (London)
[illegible]HARLES H. FAGGE (London)
[illegible]OHN FRASER, M.C. (Edinburgh)
[illegible]EORGE E. GASK, C.M.G., M.D. (London)
[illegible]. SAMPSON HANDLEY (London)
[illegible]AYMOND JOHNSON, C.B.E. (London)
[illegible] ROBERT JONES, M.V.O., C.B. (Liverpool)
[illegible] WM. ARBUTHNOT LANE, Bt., C.B. (London)
[illegible] J. LYNN-THOMAS, R.A.M.C., C.B. (Aberdeen)

Sir ALEXANDER MacCORMICK (Sydney)
Sir GEO. H. MAKINS, G.C.M.G., C.B., LL.D. (London)
WM. ERNEST MILES (London)
A. B. MITCHELL, C.B.E. (Belfast)
HENRY A. MOFFAT, D.S.O. (Capetown)
RUTHERFORD MORISON (Newcastle-on-Tyne)
Sir THOMAS MYLES, C.B. (Dublin)
CHARLES A. PANNETT (London)
R. H. PARRY (Glasgow)
Sir D'ARCY POWER, K.B.E. (London)
SIDNEY W. F. RICHARDSON (Capetown)
PERCY SARGENT, C.M.G., D.S.O. (London)
JAMES SHERREN, C.B.E. (London)
GRAHAM S. SIMPSON (Sheffield)
THOMAS SINCLAIR, C.B. (Belfast)
Sir HAROLD J. STILES, K.B.E. (Edinburgh)
GEO. GREY TURNER (Newcastle-on-Tyne)
GEORGE H. WAUGH (London)
Sir W. I. de COURCY WHEELER (Dublin)

[illegible] (Bristol), *Editorial Secretary.*

JANUARY, 1925

BRISTOL: JOHN WRIGHT & SONS LTD.
LONDON: STATIONERS' HALL COURT: SIMPKIN, MARSHALL, HAMILTON, KENT & CO. LTD.
TORONTO: THE MACMILLAN CO. OF CANADA LTD.
CALCUTTA: BUTTERWORTH & CO. (INDIA) LTD. THACKER, SPINK, & CO. BOMBAY: W. THACKER & CO.
SYDNEY: ANGUS & ROBERTSON. NEW ZEALAND: WHITCOMBE & TOMBS LTD.
UNITED STATES OF AMERICA: WILLIAM WOOD & CO., NEW YORK, *Sole Agents.*
Entered as Second-class matter at the Post Office at Woodstock, N.Y., under the Act of March 3, 1879 (Sec. 414, P. L. & R.)

The

St. Bartholomew's Hospital Operating Table

THIS table, originally designed for St. Bartholomew's Hospital, is used throughout that institution and in upwards of one hundred other Hospitals in all parts of the country. The table is a composite one and has been built on the suggestion of Mr. H. J. Waring.

A hydraulic oil-pump worked from a pedal enables the heaviest patient to be raised or lowered with but slight exertion. Rotating freely on its base, the table becomes perfectly rigid when clamped. The three ball-bearings permit of its being easily moved from the anæsthetic room to the theatre. The head and leg supports are adjustable to any angle by an automatic lever mechanism operated with one hand.

Other Fittings include:

Adjustable Back Rest
Shoulder Rests
Skull Rest **Arm Rest**
Lithotomy Crutches
Anæsthetic Screen
Instrument Tray
adjustable for any position.

Booklet giving full particulars will be sent on application.

Allen & Hanburys Ltd.

Manufacturers of Surgical Instruments and Appliances,

48 Wigmore St., London, W.1

THE
BRITISH JOURNAL OF SURGERY

VOL. XII. JANUARY, 1925. No. 47.

EPONYMS.

BY SIR D'ARCY POWER, K.B.E., LONDON.

XV. MACEWEN'S OSTEOTOMY.

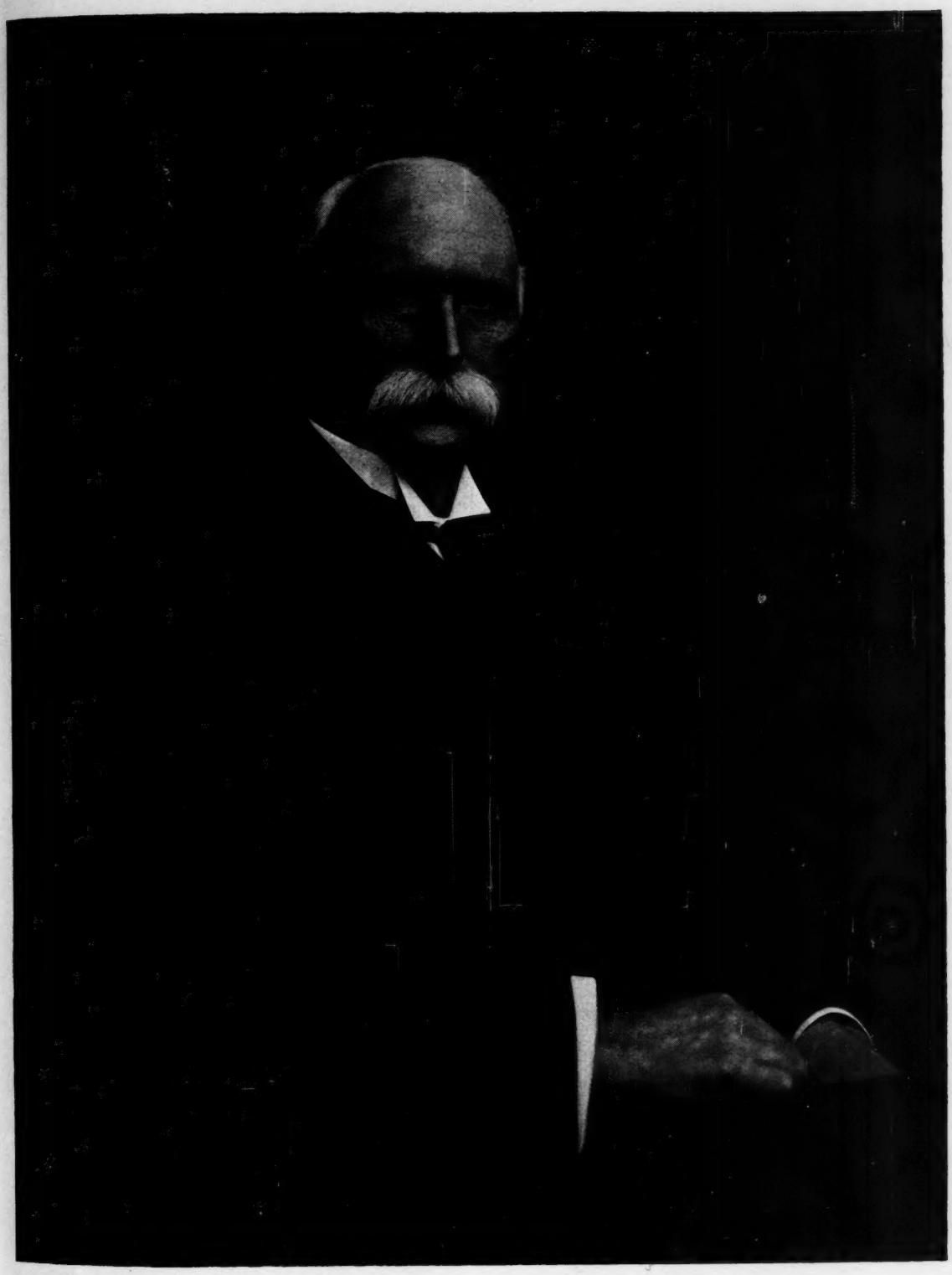
As Charles II used to call Isaac Barrow "the unfair preacher", so Macewen might be called the unfair surgeon, and for the same reason—that both were exhaustive, leaving nothing for after-comers to improve or amend. He performed his first osteotomy for genu valgum on May 19, 1877, removing a V-shaped portion from the inner side of the condyloid extremity of the femur by means of a chisel, the rest of the bone being afterwards broken. Two other patients were operated upon in like manner in the course of the year, but at some time before March 30, 1878, he had devised a method by which the bone could be divided without removing any portion of it. His attention had no doubt been drawn to the disadvantages attending the use of a chisel by a fatal case which had occurred in the practice of a surgeon who "while operating for knock-knee introduced a chisel with the straight edge downwards, which penetrated a short distance and then sloped downwards instead of going straight in. The instrument for some reason was then removed and placed on the table. Afterwards it was picked up and reinserted; this time, however, with the straight edge upwards. After traversing the aperture first made, it began to cut a course for itself and soon turned upwards, so that the incision, instead of being straight, was zig-zag. It is perfectly needless to say that such a result would be serious, and in order to obviate this tendency, another form of instrument ought to be used.

"The form most suitable for the simple osseous division is one bevelled on both sides, so as to resemble a very slender wedge. Such an instrument passes easily into the bone and maintains the direction given to it by the hand, having no tendency to glide to one side or the other. It is very easily withdrawn and does not tend to become fixed in the same way as a chisel with a shoulder such as a carpenter's. The instrument must be thoroughly trustworthy. From what I have been able to gather through others, ordinary carpenters' chisels have been used by some surgeons and they have been surprised that some of them have been broken, leaving small pieces of steel in the interior of the bone. Now the steel ought to be made to suit the

material upon which it is to act, not only in respect to its form, but also as to its temper, and the 'stuff' of which it is made. It ought to be made of very fine steel, tempered so as to prevent brittleness or too great softness. If it be brittle it may leave a portion of metal in the tissues; if too soft it will turn and curl up at the edge; of the two evils the latter is the less.

"I have had several sets of instruments manufactured for me, but the most trustworthy and satisfactory have been made by Mr. Macdonald, who put himself to some trouble to carry out my instructions concerning them and also in determining the exact temper necessary. As the latter is a matter of considerable importance the particulars arrived at may be mentioned in a couple of sentences. They were made from Stubbs' finest five-eighths steel, forged at a low heat; tempered by raising the instrument to a cherry-red heat, dipping it into oil and then plunging it into water until cold; next polished, and about an inch of the chisel, measuring from its cutting edge, having its temper drawn back by raising it to a copper-yellow colour with purple spots (probably a temperature of 550° Fahr.). In these instruments only a small portion of the chisel, embracing the cutting edge, is raised to a great degree of hardness; the remainder of the blade is comparatively soft, so as to avoid the possibility of snapping. Though this is applicable to all chisels, it is particularly so to the finest, especially when used in cases where the bone is very thick and the instrument has to be deeply inserted. A good material for testing the edge of the instrument is the hough-bone of an ox. If one finds that the instrument will neither turn nor chip in penetrating such a bone, it may be concluded that it is well-suited for cutting any human bone. These instruments stood this test. I have found them to possess the qualities of great hardness combined with toughness, and hitherto, although used on some very hard and dense human bones, they have neither turned nor chipped. The handle and the blade form one piece. The handle is octagonal, and I prefer it to the oval or cylindrical, as it gives a better grip, and enables the operator to detect more readily any deviation which the instrument may assume. The top of the chisel is furnished with a rounded projecting head against which the thumb of the operator rests as he steadies the instrument; it also serves as a support whereby the instrument may be gently levered out of the section. The figures 1 and 2 are placed near the head on the surfaces which correspond to the thickness of the blade in order that they may serve as a means of distinction when the blade is obscured in the tissues. The borders of the blade are marked with half inches, the figures being extremely light. The figures point out the depth to which the instrument has penetrated and thus serve as a guide. They are finely polished, not for appearance, but because the finer the surface the less opportunity will organic matter have of becoming adherent and afterwards decomposing.

"Now although there is nothing new in the principle on which this instrument is formed, yet its exact counterpart cannot be found out of surgery; and, though clearly of the chisel order, it has sufficient individuality to enable it to take a distinctive name. As the blade in shape resembles somewhat a transverse section of the blade of a pocket knife, and as it cuts much in the same way, it might be described as a knife for cutting bone; or, to employ a single word which would at once distinguish it from the ordinary chisel



SIR WILLIAM MACEWEN

and at the same time be more euphemistic to a patient's ear, it might be called an osteotome,"—and an osteotome it has remained to this day.

"In operating, the scalpel is introduced at once to the bone and a longitudinal incision is made a little longer than the breadth of the instrument. Through this wound the osteotome is introduced longitudinally until it comes in contact with the bone when it is turned transversely. . . . When the operator thinks the bone has been sufficiently divided, the osteotome is laid aside, a sponge saturated in 1 in 40 carbolized watery solution is placed over the wound, the surgeon holding this in one hand grasps the limb lower down and gives it a quick jerk inwards, when the bone will snap or bend as the case may be. The elastic webbing placed on the limb prior to the operation to restrain the cutaneous bleeding is now removed. As soon as the vascularity of the limb has been restored a pad of gauze is placed over the wound and the splint applied."

The whole account, which appears as a clinical lecture on antiseptic osteotomy delivered at the Glasgow Royal Infirmary in the *British Medical Journal*, 1879, i, 656–8, is of extreme interest if it be remembered that the lecture was delivered in the early spring of 1879. It shows that Macewen had invented and perfected the operation of linear osteotomy which he had performed for the first time less than a year before. It shows, too, that he had grasped the first principles of aseptic surgery, for he caused his newly invented instrument for cutting bone to be forged in a single piece and to be highly polished, when other surgeons were still using bone-handled scalpels and wooden-handled chisels. The influence of Lister is visible in the elastic webbing applied to arrest cutaneous bleeding as well as in the use of a sponge wrung out of carbolic lotion.

Sir William Macewen was born in the Island of Bute on June 22, 1848, the youngest son of John Macewen who had been in business at Rothesay and was afterwards master of the "Breadalbane", a yacht employed during the summer months to carry Free-church Ministers to and from the islands of the West Coast of Scotland. He graduated at the University of Glasgow in 1869, served as resident at the Royal Infirmary, and in 1870 became Superintendent of the Belvidere Fever Hospital. He soon resigned the post, but not until his interest had been aroused in the cerebral complications of middle-ear disease, an interest which was further fostered by Dr. Alexander Robertson, the Physician Superintendent of the Parochial Hospital. Acting as a general practitioner, as a parochial medical officer, and as a police surgeon, he had so impressed his fellow-citizens that he was appointed Visiting Surgeon to the Royal Infirmary in 1877 at the unusually early age of 29. He held this post until 1892, when he was appointed Regius Professor of Surgery in the University, and Surgeon to the Western Infirmary.

During the war he acted as Consulting Surgeon to the Navy, he operated at the Hospital at Mount Stuart, and he took a great part in establishing and carrying on the Princess Louise Scottish Hospital for Limbless Sailors and Soldiers. He was decorated C.B. for these services, having already received the honour of knighthood in 1902. In 1895 he was elected F.R.S.; in 1923 he filled the office of President of the British Medical Association, and earlier in the same year he was President of the International Society

of Medicine in succession to Dr. W. W. Keen, of Philadelphia. He died of pneumonia with all his faculties unimpaired on March 24, 1924, shortly after his return home from a visit to Australia, New Zealand, and the United States of America.

Tall, lithe, and handsome, Macewen's personal appearance was as striking as his mind was above the average. He may be placed amongst the greatest of the British surgeons not only of his own generation but perhaps of all time, both for originality of thought and for the way in which he carried his ideas into action. He was equally great as a pioneer in cerebral surgery, in the surgery of the lung, in the surgery of bone, and in those experimental branches of natural history to which he applied his leisure hours. He was fortunate in the time of his birth, for during the most impressionable years of his life, Lister was revolutionizing his branch of the profession, and the science of bacteriology was being developed. He saw the advantages of both; adopted the one with modifications of his own, and made full use of the other.

The portrait is copied by permission from a photograph by Messrs. T. & R. Annan & Sons, Glasgow.

ACUTE 'GENERAL' PERITONITIS AND ITS TREATMENT.

By W. SAMPSON HANDLEY, London.

(Being a Hunterian Lecture delivered before the Royal College of Surgeons of England on February 12, 1924.)

Though some of my conclusions may find a wider application, I shall deal in this lecture with peritonitis of appendical origin, by far the most common variety in the experience of the general surgeon.

The expression 'acute general peritonitis' is well understood clinically, as implying an unlocalized, spreading, suppurative peritonitis of grave import, unlimited by adhesions, and producing characteristic clinical signs, especially a distended, tender, rigid abdomen, with a rapidly rising pulse and a falling temperature, repeated vomiting becoming effortless in character, and cessation of the passage of flatus and fæces. So-called general peritonitis following appendicitis is very rarely universal; indeed in a twenty years' experience I can only recall one case in which the whole peritoneum was affected. This was in a boy of ten who presented no signs of obstruction and made a rapid recovery after simple drainage with removal of the appendix. The infective organisms were not sufficiently virulent to extend into and paralyse the intestinal muscle, although abundance of pus was present throughout the abdomen, even up to the dome of the diaphragm. This case incidentally helps to show that, apart from obstruction, peritonitis is not necessarily a lethal condition—a point to which I shall return.

There are few subjects in which surgery has made greater strides within living memory than in the treatment of peritonitis. Much is owing to the labours of Mickulicz, Lawson Tait, J. D. Malcolm, Treves, and others. Taking the last twenty years, the work of Dudgeon and Sargent on the pathological side, and of J. B. Murphy on the therapeutic side, is of outstanding importance. Murphy recommended removal of the septic focus if possible, drainage of the peritoneum by way of the pelvis, continuous administration by the rectum of saline infusion in large amounts, and the Fowler position during convalescence. Using these methods, Murphy was able to produce a series of 49 cases of progressive free peritonitis with only 2 deaths. The general adoption of his methods has greatly lowered the mortality, and, as was said by Mr. H. W. Carson in a recent address, they have robbed peritonitis of half its terrors.* Murphy's methods failed, however, to recognize, or at any rate to recognize fully, the importance of the intestinal paralysis which accompanies the severer forms of peritonitis. Though the Murphy methods, as Carson estimates, have lowered the mortality by 20 per cent, the residual mortality remains a considerable one (according to Gerster, 14 per cent), and

* H. W. Carson, "The Evolution of the Modern Treatment of Septic Peritonitis", *Lancet*, 1923, May 19.

this is the case even when they are supplemented by the administration of pituitrin and eserine. The use of these substances in combination is advocated on experimental grounds in a recent paper.

Sir John Bland-Sutton has recommended the intraperitoneal use of flavine solution (Browning) to arrest the spread of peritonitis, and experience has convinced me of its value. Several ounces of a 1–1000 solution may be introduced into the peritoneum without fear of toxic effects.

There are cases, however, in which all these measures fail. In some instances of streptococcal peritonitis the infection is so intense—as in one case I met with—that within twelve hours of the onset of symptoms the hands and feet are cold, the pulse running, and the patient moribund. But certain cases in which the accepted treatment fails do not belong to this hopeless fulminating variety of peritonitis; there is another group of cases, fortunately not common, which progress more slowly to a fatal issue, in spite of the most energetic treatment on accepted lines. It is to these exclusively I would ask attention in this paper, for they are cases frequently abandoned to a fate which seems inevitable, but which is in fact avoidable. The surgeon folds his hands instead of re-opening his instrument bag. He is flaccid with resignation when he ought to be taut with resolve. The characteristic feature of these cases is the failure of treatment to produce any action of the bowels, and they are dying of intestinal obstruction. The surgeon's passivity in such instances is often determined by the belief that paralysis affects the whole length of the bowel. This is not the case. The paralysis affects only certain portions, and it is amenable to treatment on the same lines as mechanical obstruction. Indeed, as Mr. Garnett Wright has insisted, the obstruction may be merely mechanical, that is to say, it may be a multiple kinking of intensely inflamed coils of bowel, glued together by plastic lymph. Perhaps more often it is due to an actual acute myositis of the inflamed bowel.

It will be necessary to trace the course of events in a case where perforation of the appendix determines the onset of an acute spreading peritonitis. In doing so I am not offering a fancy sketch, but a résumé of my experience in the operating theatre built up into a composite picture.

An area of the peritoneum of greater or less extent around the original septic focus is the first to be attacked. This zone of direct peripheral spread is, however, usually a limited and unimportant one because it can drain. Wherever the original focus is situated, the fluid exudate from this area is carried by gravity to the bottom of the pelvic cavity. Here no drainage is available. The pelvis fills from below upwards with turbid fluid, soon becoming purulent. The pelvic intestines are immersed in a bath of pus, and the peritoneal inflammation may soon extend to their muscular wall. They become intensely congested, œdematous, and thickened, and are usually contracted rather than distended. In many cases, even at this stage, they lose their muscular power altogether, and if they are thus paralysed the clinical condition is that which I described in 1914 as ileus duplex.* Often, however, it is not at so early a stage that intestinal obstruction is produced.

The first stage of general peritonitis is, then, a pelvic peritonitis. Not

---

* HANDLEY, "Ileus Duplex", *Brit. Jour. Surg.*, 1915, iii, 161.

infrequently, even now, adhesions may localize the inflammation, and a pelvic abscess results.

If, however, the inflammation remains unlocalized, the further course of events is as follows. Pus fills up the pelvis, and then extends as a rising flood among the suprapelvic intestines, inflaming and paralysing them, and producing a characteristic modification of the clinical aspect of the case. By this time, in almost all cases, and before the peritonitic flood has reached the level of the umbilicus, the intestinal paralysis is complete in the lower districts of the abdomen. From the moment when intestinal obstruction comes on, the case pursues a downhill course with increasing velocity, and the upper abdomen becomes as distended as the hypogastric region. The main toxic factor has hitherto been absorption from the peritoneal cavity, it is henceforth absorption from the distended intestines. No doubt the peritonitis continues its upward spread, aided and accelerated by intestinal distention, but long before it becomes universal, and often before it has spread much above the level of the umbilicus, the patient dies—not of peritonitis but of intestinal obstruction.

FIG. 278.—A median strip of the anterior parietal peritoneum extending from the pubes nearly to the ensiform cartilage. Below is seen on the surface of the specimen a portion of the urachus. The arrow indicates the level of the umbilicus.

The figure shows below an intense peritonitis which gradually lessens until, near the upper end of the strip, the peritoneum is almost normal. The specimen forms a kind of 'Nilometer' of the rising peritonitic flood.

It is difficult formally to prove clinical statements based mainly upon observations made in the stress of emergency operations. It has been my custom to ascertain the condition of the upper abdomen either by passing upwards from the wound a sterile swab held in forceps, or by looking up in t the cavity while the anterior abdominal wall is lift e away from the intestines by a right-angled retracto to which a vertical pull is applied. Even in 'general' peritonitis I find that the evidence of inflammation rarely extends above the umbilicus. The stomach and jejunum are distended if the case is a late one, but these and the other abdominal viscera are not inflamed. The vigorous and copious vomiting of the early stages of peritonitis shows that the stomach at any rate has not lost its peristaltic power, and the same is presumably true of the jejunum and the transverse colon. The fact that these viscera are essentially intact, though distended and embarrassed by obstruction lower down, forms the basis of successful treatment. In support of these observations I shall now give in some detail an account of the last necropsy on a case of general peritonitis which I had the opportunity of seeing.

The patient, a girl, aged 8, admitted four days after the onset of acute appendicitis, had in the interval been treated only by purgatives. Her pulse

was 140 and her condition barely justified a hasty operation. She died the following day. At the operation pus was found in the abdomen extending between the coils of intestine up to a level two inches below the umbilicus. About twelve inches of ileum, lying at the bottom of the pelvis, were glued together with lymph and dark purple in colour, with injected vessels and loss of serous lustre.

FIG. 279.—The cæcum and terminal portion of the ileum. The cæcum and the short portion of ileum lying above the pelvic brim (A to C) are slightly congested but are not acutely inflamed. No lymph is present on their surface. But the proximal ileum (A to B), which of course lay in the pelvis, is intensely congested, collapsed, œdematous, roughened, and covered with scattered patches of greenish lymph.

A, Level of pelvic brim; B, Proximal portion of ileum; C, Distal portion of cæcum.

NECROPSY, Oct. 21, 1917.—The peritoneum of the anterior abdominal wall in the suprapubic region was intensely inflamed and covered by necrotic green lymph in a thick layer. Higher up it showed intense congestion up to a level about an inch below the umbilicus. Faint congestion extended to a

FIG. 280.—The pelvic colon with the lowest portion of the suprapelvic colon. The pelvic colon (A to B) is intensely inflamed, collapsed, and thickened by œdema. The suprapelvic colon (A to C), though congested, shows a relatively mild degree of peritonitis, and the intensity of the inflammation progressively decreases from the floor of the pelvis upwards.

A, Level of pelvic brim; B, Bottom of recto-uterine pouch; A C, Proximal small portion of suprapelvic colon.

level two inches above the umbilicus. Careful inspection showed lymph on its surface three inches higher than the level where congestion ceased (*Fig.* 278). The omentum was inflamed, but when it was raised there was no congestion of the upper coils of jejunum behind it, though they were covered by pus and lymph. The upper small intestine was distended. All the small intestine below the umbilicus was intensely congested, bathed in pus, and covered with lymph.

The small intestine was now removed. Starting from the ileocæcal valve it was practically normal in colour for two inches. The next four and a half feet, representing the pelvic ileum, were intensely inflamed, coated with lymph in patches, collapsed, and thickened by œdema (*Fig.* 279). Congestion eased six feet above the ileocæcal valve. The upper eight feet of the small intestine (jejunum) were somewhat distended, but not thickened or œdematous. The upper large intestine was collapsed and for the most part congested. The pelvic colon, on the other hand, was congested, dusky, and thickened by œdema (*Fig.* 280). Evidence of the relative freedom of the upper abdomen from inflammation is further afforded by the following observations. There was no lymph on the spleen. The liver showed no evidence of peritonitis except on the right renal impression, where a little lymph was present. The surface of the stomach was free from peritonitis. There was a trace of lymph on the diaphragm, but it was otherwise normal to the naked eye.

So far I may claim to have proved: (1) That general peritonitis begins n the pelvis and spreads gradually upwards; (2) That the patient dies before the peritonitis has become universal, and often before it has reached much above the umbilicus.

What is the Cause of Death?—Sargent and Maybury maintain that the terminal event in streptococcal cases is a septicæmia, but I believe this is only true in the rarest and most acute cases, and that nearly always the proximate cause of death in suppurative peritonitis is intestinal obstruction. The obstruction is a local not a general one. The fatal dose of poison is usually absorbed from the intestine above an obstruction and not from the peritoneal cavity. Sir Cuthbert Wallace laconically stated in a recent discussion, "If the bowels can be made to act, the patient recovers; if they fail to act, he dies"—a fact which is conclusive as to the importance of obstruction, It is the predominant lethal factor in these cases.

I have already shown that if peritonitis does not produce obstruction it may become universal without being fatal. Later I shall produce evidence that if obstruction is relieved, recovery is the rule in cases otherwise inevitably fatal.

Time of Onset of Obstruction.—The time of onset of obstruction varies much. It may come on while peritonitis is still restricted to the pelvis (ileus duplex); it usually supervenes in the hypogastric stage before the peritonitis has reached the level of the umbilicus.

Position and Extent of Obstruction.—In pelvic peritonitis with obstruction, the paralysed segments of bowel are (1) the pelvic coils of ileum, and (2) the pelvic colon, hence the name 'ileus duplex', indicating the presence of two obstructions, one in the small and one in the large intestine. If peritonitis has spread up to the hypogastric region before obstruction occurs, more extensive and less defined tracts of the bowel are thrown out of action.

But as the inflammation does not extend above the umbilicus, it is safe to assume that at any rate the stomach, jejunum, and transverse colon still keep unimpaired their contractile power.

There is evidence to show that the small intestine is first paralysed, and only later the pelvic colon. Sometimes, judging by the continued passage of small amounts of flatus in cases with clear symptoms of complete obstruction, only the small intestine is paralysed; but it is never safe to assume that obstruction of the large intestine is absent. It may be added that after suitable treatment the large intestine recovers its peristaltic power earlier than the small intestine.

Prevention of Peritonitis.—Turning from the pathological to the clinical aspects of the subject, it will be convenient here to say something about the prevention of peritonitis. The avoidance of delay in operating, of unnecessary transport of the patient, of purgatives, are points that must be mentioned. At the operation it must be remembered that if peritonitis begins in the pelvis, pelvic drainage is the first essential towards its prevention. For many years I have made it a rule in all cases of appendicitis where there has been delay in calling upon the surgeon, or where mid-line hypogastric rigidity, rectal tenderness, or pain in urination suggest pelvic involvement, to begin the operation by making a small median hypogastric incision and introducing a swab to the bottom of the rectovesical pouch. In a recent case I detected thus the earliest stage of pelvic peritonitis. The pelvic intestines appeared normal except for slight distention, and so, too, on withdrawal did the swab, until closer inspection showed on its under surface a yellowish stain, about the size of a shilling, of purulent fluid. A gangrenous appendix, surrounded by adhesions, was found at the brim of the pelvis.

Those who adopt the rule of mid-line exploration and drainage of the pelvis for all cases of appendicitis not obviously early and well localized, will find collections of turbid fluid in the pelvic cavity with surprising frequency. In such cases, if the pelvis had not been drained, pelvic abscess or general peritonitis would have inevitably resulted.

It may be objected that pelvic exploration involves a risk of infecting the pelvis. This is certainly the case if the exploration is made from a right iliac incision through which an inflamed appendix has been removed, and, moreover, it is not possible to drain the pelvis satisfactorily from a right iliac incision. If, however, the exploration is median and is the first step in the operation, the risk of infection is avoided.

If the pelvis is found uninflamed and intact, the small hypogastric incision is sutured before the inflamed appendix is removed through a separate right iliac incision.

Clinical Stages of Peritonitis.—Turning now to the clinical stages of peritonitis, three stages are to be distinguished.

1. *Stage of Pelvic Peritonitis.*—Along with pyrexia, quick pulse, and cutting, constant, pelvic pain, aggravated by occasional intestinal colic, this stage is characterized by certain abdominal signs and certain signs obtained by vaginal or rectal examination. On abdominal examination there is found hypogastric distention and tenderness, but no abdominal rigidity is present, except such as may be found in the immediate neighbourhood of the original

septic focus. On rectovaginal examination, there is acute tenderness and perhaps œdematous thickening of the rectovesical fold of peritoneum and thickening of the uterosacral ligaments. These signs may be found within the first few hours, even though the primary focus, e.g., a perforated duodenal ulcer, is situated high up in the abdomen. Obstruction may or may not supervene at this stage. If it does, the condition is that of ileus duplex which I described in 1914, and which is to be regarded as an early stage of a spreading peritonitis.

2. *Stage of Hypogastric Peritonitis.*—Intervening between the pelvic stage and the typical or text-book stage of a spreading peritonitis is an intermediate stage which may be called hypogastric peritonitis, and to this I desire to direct particular attention. It is, I think, important that it should be fully recognized, for though it inevitably and rapidly passes into the third stage, it is amenable to treatment if promptly recognized. The pulse tends to rise and the temperature to fall. The patient is obviously getting rapidly worse, and is himself anxious and in extreme discomfort. Vomiting is vigorous and in considerable amounts up to two pints at not very frequent intervals, differing entirely from the frequent effortless overflow of small quantities which occurs later. It may or may not be offensive.

On examining the abdomen, the characteristic feature is the presence of distention and rigidity, tenderness, and immobility in its lower half, while above the umbilicus it is flat or only slightly distended, still soft, and only moderately tender. On palpation, a large, resonant, rounded swelling, almost as definite in its upper outline as the distended bladder and reaching about to the level of the umbilicus, can be felt in the hypogastric region. This swelling may be called the 'hypogastric football', and is doubtless formed of distended small intestine coils more or less glued together. It affords presumptive evidence of the presence of complete obstruction in the small intestine. Throughout this stage, however, small quantities of flatus may in some cases be obtained by the use of turpentine enemata, eserine, and pituitrin, and on some occasions these remedies may produce even at this stage copious evacuations, followed by the disappearance of the hypogastric swelling and the recovery of the patient, as in a recent case of my own.

The condition of the upper abdomen in hypogastric peritonitis is rather variable. It is not rigid, and still moves slightly with respiration. At first it is flat or only moderately distended, but the distention rapidly increases, and the stretching of the muscles thus produced may be mistaken for genuine rigidity.

3. *Terminal Stage.*—The hypogastric stage of peritonitis is a short one, passing perhaps within twenty-four hours into the terminal stage. The abdomen now becomes greatly and uniformly distended, for even the stomach has filled with foul fluid, and the distention extends along the small intestine as far as its inflamed segment, which is now of considerable length, since the upward spread of the inflammation has by this time involved most of the ileum. Rigidity has extended to the region above the umbilicus. The stomach begins to share in the muscular paralysis of the intestine, and the vomiting in consequence changes its character. It becomes frequent and effortless, and each time only an ounce or two of foul-smelling fluid is brought

up. It is purely mechanical and due to high pressure of fluid and gas in the stomach. This type of vomiting is exactly comparable to the escape of gastric contents from the mouth which so often occurs after death when putrefactive distention of the abdomen comes on. By the time this kind of vomiting sets in, the patient's condition is hopeless. The signs briefly are those of acute general peritonitis as described in the text-books—shrunken face, cold extremities, running pulse, and subnormal temperature; and survival is to measured by hours. This stage might fitly be termed the 'clinical picture' or hopeless stage.

Treatment by Jejunostomy.—Priority in the successful treatment of peritonitis with obstruction belongs to my colleague, Victor Bonney, whose pioneer work on the subject has received insufficient recognition. Its publication in 1910 antedated my own independent work on parallel lines. Bonney advocated the treatment of paralytic ileus due to peritonitis by the operation of jejunostomy. The operation is applicable either to the pelvic or the hypogastric stage of a peritonitis. It is an easy and a brief operation, making little demand on the patient's vital powers, and this is a great advantage. In the cases for which Bonney used it, it was very successful, but they were mostly instances of mild peritoneal infection, or non-inflammatory paralysis, following operations for the removal of infected cancers of the cervix. Even if it had been proved that the operation would be equally successful in the intense peritoneal inflammations following appendicitis, the objection in my opinion remains that not only does jejunostomy necessitate a second operation within a fortnight for the resection of the jejunum, but that meantime, by reducing the available absorptive surface, it robs the patient of much-needed fluid and nutriment at a time when he urgently requires all he can absorb.

Treatment by Ileo- or Jejuno-colostomy combined with Cæcostomy.—My own aim in the treatment has been to construct, above the level of the peritonitic flood, a complete emergency alimentary canal, providing a considerable area of absorptive surface. I have compared this policy with that of a man whose house is invaded by a rising flood. He must leave the ground floor and make temporary provision on the first floor for all the primary needs of life until the flood subsides.

Treatment in the Pelvic Stage.—If obstruction comes on in the pelvic stage of peritonitis (ileus duplex) and fails to yield to treatment, the right course in my opinion is to do an ileocæcostomy and to tie a catheter into the cæcum. This method has proved uniformly successful in my hands, though in five earlier cases treated merely by appendectomy and peritoneal drainage four deaths occurred, and in seven cases treated by ileocolostomy alone five deaths took place. This stage is shown in *Fig.* 281.

Treatment in the Hypogastric Stage.—If obstruction occurs in the hypogastric stage of peritonitis, it must be assumed that the whole of the ileum is inflamed and paralysed, and probably also the sigmoid flexure and pelvic colon. Here my plan has been to select a distended but uninflamed coil of the jejunum and to anastomose it either to the transverse colon or the ascending colon, it matters not which. A Paul's tube is then tied into the cæcum (*Fig.* 282).

During the last nine years, out of a large number of operations for acute

appendicitis, I have met with four cases of general peritonitis in the hypogastric stage, all of them infections of the gravest type. In all of them the usual treatment of appendectomy, pelvic drainage, the Fowler position, saline infusion, and the administration of eserine, pituitrin, and purgatives was perseveringly tried. In all of them, nevertheless, the patient had reached a

FIG. 281.—Ileus duplex arising from gangrenous pelvic appendicitis. The pelvis is occupied by a pool of pus bathing the inflamed pelvic ileum and pelvic colon. The last two inches of the ileum is normal. The supra-pelvic ileum is much distended, but is neither paralysed nor inflamed. A fairly sharp line separates it from the inflamed portion of the ileum. There is no peritonitis above the pelvic brim.

condition absolutely hopeless according to my previous clinical experience. They were all dealt with on the plan I have outlined. The abdomen was reopened and a jejunocolostomy followed by a cæcostomy was done as rapidly as possible with a minimum of handling of the intestinal contents.

In all four cases the operation was followed within forty-eight hours by softening and subsidence of the abdomen, the cessation of vomiting, and the return of natural evacuations per anum. The peritonitis ceased to spread and the patient's general condition rapidly improved. In three of the cases complete recovery occurred with spontaneous closure of the cæcal fistula. In the

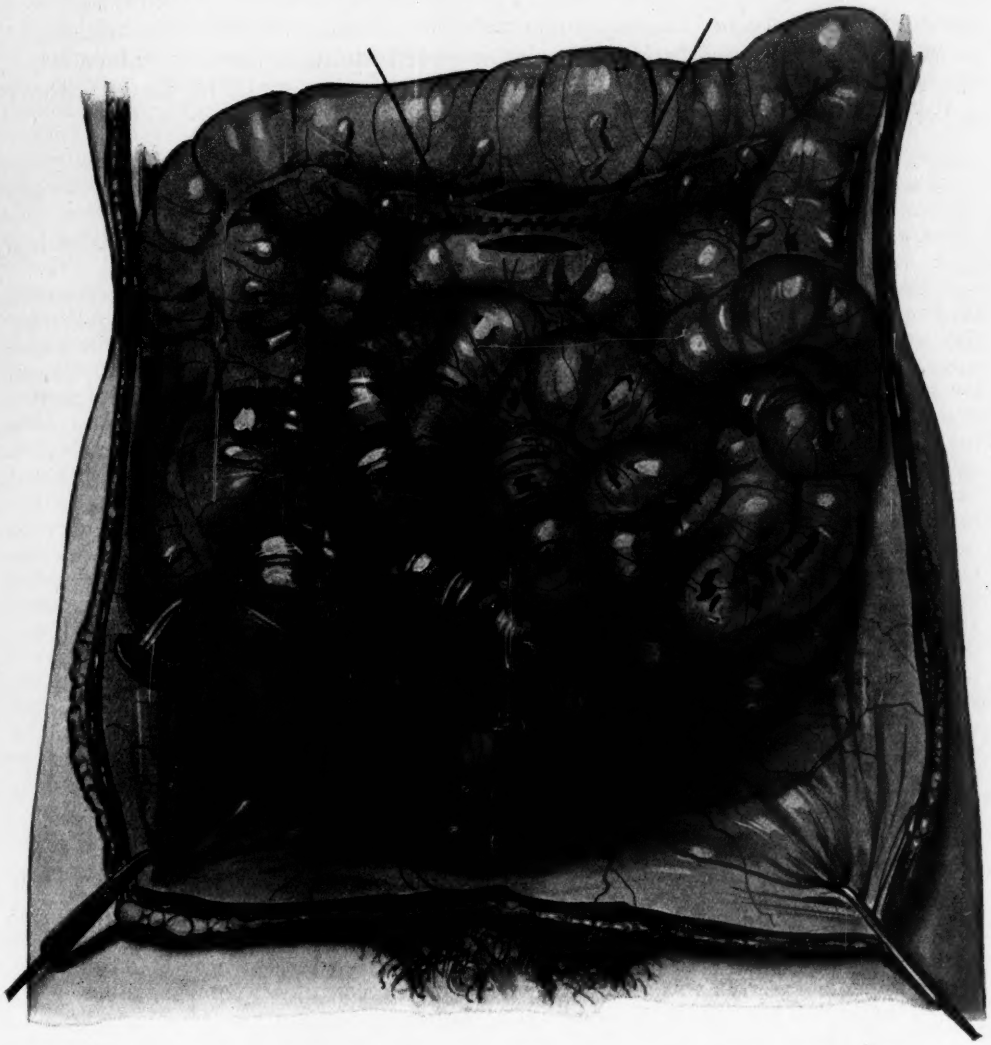


FIG. 282.—Hypogastric peritonitis with obstruction and its treatment by jejunocolostomy combined with cæcostomy. Note that the peritonitis diminishes from below upwards, and that the intestines above the umbilicus, though much distended, do not seem sufficiently inflamed to impair their contractile power.

fourth case the streptococcal infection was so intense that although the peritonitis subsided and normal bowel actions returned, the patient died a month later of typical pyæmia with multiple abscesses.

It is upon the clinical evidence of these four cases that I rely in the main

to show that death in appendical peritonitis is really due to intestinal obstruction. Remove that factor and the patient recovers. The unsuccessful case is the most instructive of all. It shows that if intestinal obstruction be averted, peritonitis as such is a recoverable disease, even if the peritoneal infection is so intense as to end in pyæmia.

The treatment of these very acute cases of general peritonitis makes demands not only on the surgeon's initiative and technical skill, but on his moral courage and resolution. If the accepted methods fail to produce relief, the painful necessity of advising a second operation must be faced without delay. Only thus can a disaster be averted.

ILLUSTRATIVE CASES.

Case 1.—Miss H., age 30, had for some months complained of vague abdominal pain at intervals, but had not sought medical advice.

On Dec. 29, 1915, she was taken ill with what was supposed to be a chill on the liver. She kept at her work, and did not consult a doctor. The following day vomiting began, and was repeated soon after she took a purgative. The abdominal pain, of a colicky character, was situated in the lower part of the abdomen. On Dec. 31, she went to town as usual, but was sick and ill, and the pain persisted. Jan. 1, she was worse, and was then first seen by Dr. A. J. Hogg, of Ealing. He found that she had general severe abdominal pain and tenderness, worse on the left side. There was little movement of the abdomen on breathing. The vomiting had been bilious, and the temperature was 100°, the pulse about 90. At 6 p.m. on the same day the pulse was quicker and the temperature higher. There was great pain in the lower part of the abdomen, and she lay on her back with her legs drawn up. On this day for the first time there was pain on micturition, and in the evening Dr. Hogg asked me to see her with him with a view to operation. About 10 p.m., when I saw her, her pulse was 120, her colour good, and she did not appear to be in much distress. There was slight general abdominal distention, and the abdomen moved to a certain extent with respiration. There was no true abdominal rigidity. In the right iliac fossa an indefinite resistance could be felt. On pelvic examination, there was extreme tenderness in the region of the pouch of Douglas, with fixation of the uterus, thickening of the right uterosacral fold, and some resistance in the right broad ligament. The periods were regular, the last one having ceased a fortnight previously. The patient's statements as to the passage of flatus were rather vague, but she did not appear to have passed it more than once in the preceding twenty-four hours.

In view of the early colicky hypogastric pain, of the signs on the right side of the pelvis, and of the pain on micturition, together with the absence of abdominal rigidity and the presence of resistance in the appendix region probably due to swollen ileocolic glands, we arrived at the conclusion that the patient was suffering from pelvic appendicitis. It was thought, in view of the continued vomiting, that intestinal obstruction was likely to complicate the case.

First Operation.—Immediate operation was decided upon, and a median hypogastric incision was made. On opening the peritoneum a certain amount of clear fluid escaped. The omentum was adherent in the pelvis, and on separation it was found to be sloughing at its tip. The coils of small intestine, which now came into view, were rather congested, but had not lost their lustre. The pelvis was packed off from the rest of the abdomen, and on passing the hand down into it, some ounces of thick pus escaped, and a mass was found which, on extraction, proved to be a swollen and gangrenous appendix containing two large concretions. There was superficial sloughing of a portion of the cæcum near the base of the appendix. All the intestines in the lower abdomen were distended by flatus and rather congested, but there were no areas of œdematous, collapsed, and deeply-congested intestine such as are seen in typical cases of ileus duplex. The condition appeared

to be general paresis of the pelvic and suprapelvic intestines without any actual paralysis, and it seemed likely that eserine and pituitary extract would maintain peristalsis in the damaged gut, but it appeared safer to tie a catheter into the cæcum and bring it out through the median wound. The pelvis was drained by two large tubes.

After the operation the patient was placed in the Fowler position, saline was administered subcutaneously to the extent of about six pints for the first twenty-four hours, and injections of eserine salicylate were given every six hours in order to produce intestinal contractions. The catheter, either because it was blocked, or because there were no contractions in the intestine, did not act. Turpentine enemas were given, but only produced a little fæcal-stained fluid, and no flatus passed per anum. A purgative failed to act, and forty-eight hours after the operation the condition of the patient was most alarming. The abdomen up to the umbilicus was rigid, hard, and tensely distended like a football. Above the umbilicus the distention was not so marked; rigidity was absent, and there was some abdominal movement with respiration. The pulse had risen in frequency. The patient was not sick, but was troubled by constant feelings of nausea which made her unwilling to take nourishment.

Second Operation.—Since I have never seen a patient recover from a similar condition of general peritonitis with complete obstruction, I told the friends that recovery was improbable in any circumstances, and certainly impossible unless something further was done. It appeared certain that the peritonitis had extended as high as the level of the umbilicus, and that the whole of the ileum and the lower colon were suffering from paralysis, the result of the peritonitis. The condition of the abdomen above the umbilicus showed, however, that the upper part of the small intestine—say, roughly, the whole of the jejunum—was still capable of normal peristalsis. Though the ascending and descending colon were probably paralytic, the transverse colon lying at the upper limit of the peritonitic area might be presumed still to retain its contractile power. A lateral anastomosis between the lower jejunum and the transverse colon seemed, therefore, to be the rational method of treatment if combined with a cæcostomy to afford a free exit for the contents of the large intestine.

The friends of the patient consented to further operative measures, and through an incision 4 in. long situated 1½ in. to the right of the middle line and extending downwards to an inch below the umbilicus, I re-opened the abdomen.

The incision exposed the lower border of the transverse colon, which was distended by flatus and apparently not inflamed. Just below the transverse colon I made a small opening in the great omentum, and thus obtained access to the upper small intestine. The first coils which presented (belonging probably to the upper ileum or lower jejunum) were too much distended and inflamed to permit of their use for the lateral anastomosis—a proof that the peritonitis had actually extended up to the umbilicus. Two fingers introduced in a direction upwards to the left succeeded in withdrawing a higher coil, which though distended was not inflamed. This coil was brought forward in the opening in the great omentum and clamped. A clamp was next applied to the closely adjoining portion of the transverse colon, which also had been brought out into the wound. The area was packed off, and a lateral anastomosis was made in the usual way. Since the small intestine, owing to distention and slight œdema, did not hold stitches well, a portion of the great omentum was drawn down over the suture line so as to strengthen it. The bowel was returned and the abdominal incision was sewn up. The cæcum was next sought in the original wound at the point where the catheter was tied in it, and here a Paul's tube was inserted.

After-History.—The patient bore the operation well, and her pulse at the end of it was about 120. It should be added, as indicating the virulence of the bacteria present, that the whole of the surfaces of the original incision were at this time covered by a superficial grey slough. The result of this operation was most satisfactory. It was followed by the administration of a purgative, and the following day the cæcostomy had acted well. A certain amount of fluid material together

with flatus had also passed by the anus. The lower abdomen, though still tender, had lost its tension, and the rigidity was less marked. Three days after the operation a tender swelling developed in the right iliac region. There was also some suppuration of the incision of the second operation. The right iliac swelling was opened two days later, and proved to be an abscess in the abdominal parietes, probably due to extension of infection from the region of the second operation. No leakage took place at the lateral anastomosis. When the Paul's tube came out, there was a considerable escape of semi-fluid intestinal contents from the cæcostomy, and the bowels also continued to act per anum. The patient's temperature fell, the wound assumed a healthy aspect, and her appetite returned. For some time after the operation a fluid action of the colotomy wound followed each meal. Nutrition rapidly improved and strength returned. The cæcostomy wound contracted and closed.

The anæsthetics were given by Mr. Herbert Charles. I am indebted to Drs. A. J. Hogg and W. B. Stanford for their unremitting care in the after-treatment, which alone rendered recovery possible.

The patient remained well for years afterwards, but was reported in 1924 to present signs of pernicious anæmia.

Case 2.—Mr. B. B., an actor, age 24, had been ill for a fortnight in 1917 with symptoms at first thought to point to appendicitis, but later considered to be due to pneumonia on account or signs at the right base. On the tenth day of his illness he was moved from Southend and placed under the care of the late Dr. Wright, of Warwick Crescent. On the following day his temperature descended by crisis, but on the thirteenth day he complained of pain in the right iliac fossa and Dr. Wright noticed a lump there. The next day he was worse, and I was asked to see him. He was in good general condition with a pulse of 84 and a temperature of about 100°. The abdomen was soft and moved on respiration, but in the right iliac fossa there was a large definite lump extending upwards and backwards into the right lumbar region. Dr. Wright and I agreed that an appendix abscess was present, and the patient was operated upon the same evening. A large quantity of fœtid pus was evacuated through an incision in the right iliac region. The abscess cavity was sloughy and extended upwards in the right loin as far as the liver. A counter opening was therefore made in the right loin. The appendix could not be found and had apparently sloughed away. A prolonged search for it was not considered advisable. The cavity was washed with normal saline and with flavine, 1–1000. The following morning at 6 a.m. he had passed urine and had not vomited, but flatus had not passed. He was perspiring freely. At 6.30 p.m. the day after operation, he vomited for the first time, and this was repeated frequently during the succeeding night. On Oct. 1, a turpentine enema and injections of eserine and pituitary were unsuccessful in causing the passage of flatus except in the smallest quantities, and although his temperature had fallen practically to normal, intestinal obstruction was evidently present. On Oct. 2, the abdomen below the umbilicus showed rigidity and a football-like distention, while above the umbilicus it was somewhat distended but not rigid. It was obvious that the patient would die of obstruction unless relieved, and at 2 p.m. on the same day I again operated through a median hypogastric incision. On passing the hand into the pelvis some turbid fluid escaped, and near the bottom of the pouch of Douglas several loculated abscesses containing thick pus were evacuated. Two large drainage tubes were inserted into the pelvis. The condition of the intestines was now investigated. The cæcum was found to be collapsed and almost normal in appearance, but the small intestines below a horizontal line drawn two inches beneath the umbilicus were congested, inflamed, and distended, and sharply kinked in many places by sticky lymph. It was evident that there was a diffuse paralytic obstruction up to the lower part of the small intestine. The incision was prolonged upwards as far as the transverse colon, which was found normal and collapsed. The small intestine in this neighbourhood though distended was not inflamed or œdematous. It was, however, so much distended that immediate relief appeared imperative, and through two small punctures made with a knife and subsequently closed with two purse-string sutures a considerable length of the

jejunum was partially emptied. A coil of distended jejunum was selected above the inflammatory area, brought through a hole made in the great omentum just beneath the transverse colon, and anastomosed rapidly to the transverse colon. Since the pelvic colon was presumably inflamed and obstructed, it was now necessary to provide drainage for the large intestine. The incision made at the first operation was opened up and a large catheter was tied into the cæcum. An evil-smelling, brownish fluid flowing almost at once from the catheter indicated that the small intestine was relieving itself into the transverse colon and thence into the cæcum.

The following day there was a most remarkable improvement in the patient's condition. Instead of presenting the distressed aspect of a dying man, he was comfortable and had lost the expression of anxiety so noticeable the day before. His pulse had fallen in rate, and, instead of being distended, the abdomen was quite flaccid, though still rather convex. Vomiting had ceased and did not again recur. The temperature, however, remained high, and on Dr. Wright's suggestion, antistreptococcus serum was admininstered with apparently excellent results. The catheter came out of the cæcum on the fifth day, but even before this time the bowels had acted normally. About three weeks after the operation a recurrence of pyrexia with rapid pulse and cardiac distress was apparently caused by a residual abscess at the bottom of the pelvis. This was treated by hot rectal injections of water and ruptured into the rectum.

At the present time (1924) the patient is, I believe, quite well.

Case 3.—On the night of March 13, 1918, Mrs. J., age about 30, was seized while in bed with a sudden acute abdominal pain. Dr. J. J. Edwards was called in and found some rigidity of the right rectus muscle. The temperature was normal and the pulse not much raised. He diagnosed appendicitis. The following morning he asked me to see her. I found the abdomen slightly and uniformly distended. There was marked tenderness of the right iliac fossa and here a definite resistance could be felt. The lower part of the right rectus muscle was distinctly rigid. Immediate operation was advised, and was carried out as soon as the patient could be removed to a nursing home. An incision in the right iliac fossa showed free pus surrounding the cæcum. The rest of the abdomen appeared to be free of infection, but there were no barriers of any sort to impede the pus. The appendix was rather swollen and rigid, but was not distended or strictured. The cæcum was greatly distended by flatus, and much congested. I was in doubt whether the case was appendicitis or typhlitis. Either diagnosis seemed possible, but in any case the bacterial invasion of the abdomen had not occurred through a gross perforation. As infection of the pelvis seemed to be a certainty, a small opening was made above the pubes and a drainage tube introduced into Douglas' pouch. The Fallopian tubes were congested, but otherwise normal, and there seemed no evidence of ascending genital infection. The following day the bowels had acted and flatus had been passed as the result of turpentine enemas, but below the umbilicus the abdomen was distinctly swollen and the lower halves of both recti were rather rigid. Injections of eserine and pituitary extract were ordered. No vomiting had occurred, but the pulse was more rapid than on the previous day. An injection of antistreptococcus serum was given. The day following the patient seemed more toxic; she was rather drowsy and apathetic, and a large dose of serum was administered, apparently with great benefit. A cultivation of the pus had shown the presence of the streptococcus. The distention below the umbilicus was more marked, but vomiting still remained absent. A day later the sub-umbilical distention had increased still further, and now conveyed the impression of a half-inflated football in the lower part of the abdomen. The epigastric region was convex but soft. The patient vomited several times. It was evident that almost complete paralytic obstruction was present. The pulse had increased to 120. She was seen with me by Dr. Campbell Thomson, who agreed that her condition was hopeless unless something further was done. The abdomen was reopened on March 19, and almost the whole of the small intestine was found greatly distended, pressing the transverse colon upwards and forwards so that it could not be reached. The ascending colon was drawn out from the wound, and a distended

coil of jejunum was anastomosed to it. A small Paul's tube was now tied into the cæcum.

At the end of the operation the patient's condition was very grave and her pulse 140. The anæsthetist, when he saw me the following day, refrained from asking about the patient because he felt sure she could not survive the night. However, the following morning—March 20—her condition was rather better. During the thirty-six hours following the operation, 36 oz. of fluid were passed by the cæcal tube. On March 21, the improvement was great; the abdomen was quite soft though still distended below the umbilicus; above the umbilicus it was flat. The pulse, though rapid (140), was full instead of being small and feeble as on the previous day. On this day for the first time she began to take food in quantity; 4½ pints of fluid nourishment were administered by the mouth during the day. At night she was a little toxic and drowsy. About 20 oz. of urine were passed in the twenty-four hours following operation. The temperature was normal. On March 22, she was much troubled by hiccup; the abdomen was soft and wrinkled. Her morale, which had been extraordinarily good, went to pieces about this time; she was tired and was inclined to give up the struggle. She only recognized friends slowly and with difficulty, and asked for morphia although she was not in pain. During this day she passed about 44 oz. of urine. The force of the poison on the viscera appeared to be exhausted, but it was now playing havoc with her nervous system, upon which in the early days it had seemed to act as a stimulant. The temperature was steady on the normal line and she took food well, but the pulse remained between 130 and 140. Its rapidity in proportion to the temperature was a characteristic feature of the case all through. On March 23 there was incontinence of urine and fæces. The patient was drowsy and did not recognize visitors, lying with eyes half open in a state of semi-coma. The tube continued to act well and the abdomen was now perfectly flaccid though still convex; it moved freely on respiration. Œdema of the vulva had appeared, probably due to intense pelvic peritonitis and consequent œdema of the pelvic cellular tissues. Three pints of fluid were passed by the cæcal opening and 3½ pints of fluid were taken. On the evening of the same day, the patient was quite clear in her mind, with a strong voice and a better pulse (128). The anus had this day acted more than the tube, and had passed partly-formed motions. The abdomen, everywhere flaccid, was slightly concave above the umbilicus and slightly convex below it. The abdominal stitches were irritating and cutting through. The vulva œdema had nearly gone. No incontinence of urine; tongue moister. There was marked painful swelling of the right parotid gland. On the 24th the improvement was maintained. Four loose motions were passed per anum and very little fluid was drained away from the cæcum. A right parotid abscess was opened and washed out with flavine. On the 26th the Paul's tube was removed and the fistula plugged with gauze. The patient was comfortable, and was taking food well. Temperature normal, pulse 128. The same day there was some secondary hæmorrhage from the abdominal wound which weakened her considerably. On the 27th, under an anæsthetic, the cæcal opening was closed and the abdominal wound re-sutured. Owing to intense local infection, all the old stitches had cut through and the wound was gaping widely. After the operation she was much collapsed, and for several days her condition was very critical, but she again rallied. On the 28th the lower part of the abdomen was found to be distended. Owing to a misunderstanding, the flatus tube had not been regularly used subsequent to the closing of the cæcal fistula. On April 1, she had rallied enough to enjoy solid food such as chicken, and she smoked a cigarette although still in an exhausted condition. Later in the day a profuse fæcal discharge took place from the abdominal wound, owing to the breaking down of the cæcal opening. This was a very unfortunate event, and gave rise from this time onwards to great trouble. On April 2 she complained of pain in the right elbow. On the 3rd some hæmorrhage took place from the bowels: this was repeated on the 4th. On the 5th the abdominal wound was looking cleaner and granulations were forming on its edges. The stitches were again cutting through. The wound was strapped across to hold it together. On about April 5, she developed a cough with some purulent expectoration, but no signs of empyema could be detected. She continued to take food well. On the 7th she

was still taking solid food and again enjoyed a cigarette ; pulse 128. On the 8th it was noted that the abdomen was perfectly flat and soft—if anything retracted, and the wounds were beginning to granulate. A swelling was noted on the right shoulder and was incised. It contained pus but did not communicate with the shoulder-joint. The nurse notes that she had taken food well and seemed very bright in the evening. She had a good night and slept well. On the following morning (the 9th) she felt tired out and seemed very weak, although she continued to take food well. From this time she began to go downhill rapidly from wasting and exhaustion. On the 14th a fluctuating swelling was detected to the left of the 4th dorsal vertebra : at this point the spine showed an angular change of direction due either to a pyæmic abscess in the vertebra or to the lighting up of an old spinal caries. A fluid swelling appeared in the sacral region, evidently another abscess, her lips were blue, and there was much bronchial secretion which she could not get up without painful effort. She sank into a state of semi-consciousness and died on April 15.

Case 4.—Mr. M., age 24, on May 8, 1923, felt discomfort and nausea during the day, and vomited four or five times during the night, but on the next morning two pills he had taken acted well, and he ate scrambled eggs for breakfast. Dr. W. A. Rudd saw him at 11 a.m., and suspected appendicitis. An hour or two later terrible right iliac pain suddenly came on and got rapidly worse. When I first saw him, about 2 p.m. on the same day, there was acute deep tenderness in the appendix region, a raised temperature, a pulse of over 100, and abdominal rigidity in the right iliac and lumbar region. Retrocæcal appendicitis was diagnosed, and at 4 p.m. the abdomen was opened by a small median hypogastric incision. The pelvis was found to be full of pus, and a large drainage tube was introduced into the rectovesical pouch. The appendix was then removed through a right iliac incision. It was swollen and intensely inflamed, and presented a patch of gangrene. The pelvic intestines, though inflamed, presented no evidence of paralysis. Nevertheless, the onset of paralytic obstruction was feared, on account of the repeated vomiting. Continuous saline was given per rectum. Pituitrin, 1 c.c., was given every four hours, and also several turpentine enemas. Flatus passed only in very small quantities until the 11th, when a turpentine enema produced a good result and gave much relief. At this time the temperature was 99·2° and the pulse 78. On the 12th calomel was given hourly in $\frac{1}{10}$-gr. doses for twelve hours. Flatulence was troublesome, and he vomited for the first time since the operation. During the early morning of the 13th he vomited copiously a brownish fluid, and a turpentine enema evacuated only a very small amount of flatus. Hiccup came on occasionally and became continuous. Distention increased and vomiting returned and became bilious. He was restless and uncomfortable. The pulse rose to over 100, and the temperature was slightly subnormal. Late on the evening of the 13th, when I saw him again, he presented a typical picture of hypogastric peritonitis. The abdomen was rigid up to the umbilicus; above this, though the muscles were stretched from distention, there was no rigidity. On deep palpation a firm but elastic football-like mass seemed to occupy the central hypogastric region, while above this level the intestines were distended. The pelvic tube was draining freely and the bladder was empty. It was evident that his condition was desperate. Two pints of offensive brownish fluid had been vomited during the evening. At 2 a.m., under light anæsthesia, the abdomen was re-opened just to the left of the umbilicus, and search was made amid distended but uninflamed coils of jejunum for the transverse colon. It appeared to be fixed high up in the abdomen and could not be reached. The right iliac incision was therefore re-opened, and an uninflamed coil of jejunum was brought down and anastomosed with the cæcum, into which a Paul's tube was subsequently tied. The operation caused serious shock, but at 6.30 a.m. the temperature was 98·8° and the pulse 102. The distention of the abdomen was relieved within twelve hours of the operation, but for two days the patient's general condition gave rise to great anxiety. The tube in the cæcum was repeatedly blocked, and was kept clear by injections of saline, sometimes combined with Epsom salts. He vomited a brownish fluid twice on the 14th. Temperature 99·8°, pulse 132, colour bad, some delirium. During the night of the 14th the bowels acted, the stool was liquid and

light brown in colour; vomiting continued. On the 15th the bowels were freely open, and from this date to the 17th there were repeated large liquid actions, though very little drained from the cæcal tube. Subsequent improvement was rapid and continuous, the cæcostomy closed spontaneously, and he left the home with his wounds healed on June 24, 1923. His present condition (Jan., 1924), is satisfactory, and there is no undue looseness of the bowels.

In this case the absence of absolute constipation before the anastomosis, the promptitude with which loose actions per anum began after it, and the smallness if the discharge from the cæcal tube, show that the pelvic colon had not shared in the complete paralysis of the pelvic and hypogastric small intestine. This was not altogether an advantage, for it involved the risk that the empty and thirsty colon might absorb a fatal dose of toxins from the septic contents of the obstructed small intestine which reached it via the anastomosis. Between the 14th and the 17th, while the bowel was emptying itself, toxic symptoms were prominent and the pulse attained a maximum of 135. The pulse-rate subsequently fell. It remained at about the rate of 100 until May 29, and fell to 80 before he left the home.

THE CHOLESTEROL CONTENT OF THE BLOOD IN RELATION TO GENITO-URINARY SEPSIS.

By WILLIAM MacADAM and CECILIA SHISKIN, Leeds.

In the surgical treatment of cases of obstruction in the lower urinary tract, especially that due to prostatic enlargement, there are two aspects of the patient's general condition which have to be considered in deciding on the nature of the operative procedure required, and on the most favourable time for its successful performance. These are :—

1. *The Functional Efficiency of the Kidneys.* Defective renal function may be either of a permanent type due to gross interstitial and glomerular changes in the kidney, or of a temporary nature due entirely to the effects of mechanical back-pressure.

2. *The Capacity of the Patient to Resist the Spread of Sepsis in or to the Upper Urinary Tract and Renal Parenchyma,* since a local focus of infection in the form of a cystitis of greater or less degree is almost invariably present in these cases.

1. As regards the estimation of defective renal function, it was the consensus of opinion in the recent Discussion at the Royal Society of Medicine[1] (1922) that functional tests, and especially the estimation of the blood-urea nitrogen, do give some real information—conclusions which were in keeping with the results of an investigation previously carried out by Dobson and one of us (W. MacA.)[2]. From the surgical point of view it was concluded that the blood-urea estimation is a reliable guide to the functional capacity of the kidney ; that its estimation should be a routine procedure in every case of enlarged prostate before operation ; and that where there is evidence of nitrogen retention, prostatectomy should be postponed until the blood-urea has become normal as the result of suprapubic drainage or other treatment.

2. But the second question of the possible spread of sepsis to the kidney seems to be even more important, and it is one in regard to which clinical examination alone often fails to give a satisfactory answer. To quote from the remarks of K. M. Walker[3] at the discussion on tests of renal function: "Most of us appear to have been let down at one time or another by our functional tests, and it would seem that there exists a factor which we have at present no means of estimating, when assessing the risks of an operation. I am convinced that sepsis is the factor that so frequently upsets our calculations and defies our tests. Many patients who are said to have died of renal inefficiency after prostatectomy have, in my opinion, died rather of sepsis superimposed on a damaged kidney . . ."

For some time we have devoted attention to the elaboration of a test which might be of practical value in estimating this factor of sepsis, especially in cases where the clinical signs are few or uncertain.

The tendency to a condition of acidosis in cases of infection[4] suggested a possible means of determining the presence of a pyelitis or pyelonephritis in genito-urinary surgical conditions, but our investigations into the changes in reaction and alkali reserve of the blood in cases of prostatic enlargement led only to equivocal results. We then turned our attention to the possible value of estimations of the cholesterol content of the blood, and the present paper is an account of our findings.

CHOLESTEROL METABOLISM IN RELATION TO IMMUNITY.

Both experimental research and clinical observation seem to show that cholesterol plays an important rôle in the processes of immunity. It is said to stimulate the production of antibodies, but its precise function is still undefined. Chauffard demonstrated that severe infections are always accompanied by a marked hypocholesterolæmia, while Landau[5] first pointed out that the suprarenal cortex shows a very low content of lipoid material in patients who have died of bacterial and especially pyogenic infections.

On the other hand immunization processes have been shown to be accompanied by an exceptionally high cholesterol content of the blood. An attempt has been made by Barbary[6] to make some practical application of this knowledge concerning one of the functions of this important lipoid. By injections of cholesterol in combination with camphor, which he believes stimulates the production of cholesterol, there is a rapid increase of the cholesterol content of the blood; and in the case of soldiers suffering from septic wounds he found that the mortality was diminished and the period of convalescence shortened as compared with control cases which received no such injections.

SCOPE OF INVESTIGATION AND METHODS OF PROCEDURE.

It was decided to investigate the cholesterol content of the blood in a series of cases of urinary obstruction due to enlarged prostate—where, of course, there is usually some degree of cystitis—in order to determine whether such an investigation might afford any evidence of the amount of resistance to the spread of infection, and of the possibilities of 'surgical kidney' or other form of renal sepsis supervening.

Eighty-eight cases of prostatic obstruction confirmed by operation or post-mortem examination have been examined. In the course of the investigation many other genito-urinary conditions, such as urethral stricture, vesical calculus, papilloma of the bladder, renal hypernephroma, etc., have been examined incidentally, but our protocols bear reference only to cases of prostatic enlargement.

Blood was obtained by venous puncture before any operative interference, whether suprapubic drainage or prostatectomy, was carried out. This is of considerable importance, because, as described below, a marked fall in blood-cholesterol is an almost invariable sequela of any operative procedure.

The urea nitrogen of the blood was also estimated in all cases in order to compare the relative incidence and importance of nitrogen retention and hypocholesterolæmia in genito-urinary obstruction.

The estimation of cholesterol was carried out by a slight modification of Myers and Wardell's method, as described by us[7], while the urea nitrogen was estimated by Folin and Wu's method[8].

THE BLOOD-CHOLESTEROL OF NORMAL PERSONS AND ITS VARIATION WITH AGE AND OTHER FACTORS.

Using this method of Myers and Wardell, we found the cholesterol of the blood plasma in a series of twelve normal cases—all except one under 50 years of age—to vary between 0·191 per cent and 0·133 per cent, with an average of 0·161 per cent. These values are in agreement with those of other workers, e.g., Chauffard, Laroche, and Grigaut[9], whose figures are 0·150 per cent to 0·180 per cent. It has to be remembered that, as all our cases are over 50 years of age, the average normal cholesterol content should be reckoned as somewhat higher, since a hypercholesterolæmia is commonly associated with arteriosclerosis, granular kidney, and other degenerative conditions of the later periods of life.

Analysis of Results.—Of the 88 cases there were 14 of malignant prostate, and the cholesterol findings in these will be discussed separately. Of the 74 cases of benign enlargement of the prostate, prostatectomy was carried out in 56, either as a single or two-stage operation. The remaining 18 were considered unsuitable for major operation, sometimes on clinical grounds alone, in many instances as a result of our laboratory investigations. This applies especially to the later cases, when increasing confidence in the value of the test allowed us to express an opinion as to the operative risk of prostatectomy. Hence in 13 cases, suprapubic drainage alone was carried out, while in the remaining 5, catheterization was resorted to.

Of the patients, 27 died, and an autopsy was obtained in all except four. It has thus been possible to correlate fully our findings with the degree and distribution of sepsis present.

It should be noted that there has been no selection of cases. The great majority were hospital patients of the Leeds General Infirmary, many being admitted as urgent cases of urinary obstruction, and practically all suffering from a greater or less degree of cystitis.

ANALYSIS OF CASES HAVING A LOW BLOOD-CHOLESTEROL. (*Table I.*)

As mentioned above, the average normal cholesterol content is 0·160 per cent, with a range of variation from 0·130 per cent to 0·190 per cent. From an analysis of the cholesterol findings in the present investigation, we have concluded that all values below 0·130 per cent may be taken as abnormally low, and signify that the patients possess little resistance to bacterial infection, and are poor 'operative risks'.

Thus, out of the total series of 88 cases, 18 gave a cholesterol content less than 0·126 per cent. Of these 16 died of pyelonephritis, or other form of renal sepsis, and 2 recovered. From the clinical standpoint, only 10, or just over 50 per cent, were considered bad surgical risks, and it is noteworthy that 8 of these were suffering from marked renal inefficiency, as shown by their having a high blood-urea content.

Prostatectomy was carried out in 5 of these patients, and 3 died. Of the others, suprapubic cystotomy was performed as a measure of relief in 7 cases, while the remaining 6 were catheterized.

Table I.—Analysis of 18 Cases with Low Blood-cholesterol (below 0·130 per cent), with 16 Deaths and 2 Recoveries.

Case No.	Clinical Condition	Operative Treatment	Blood		Results and Post-mortem Findings
			Cholesterol	Urea Nitrogen	
3	Poor	C	0·089 grm.	25 mgrm.	Death. Pyelonephritis
10	Fair	C	0·126 ,,	75 ,,	,, Multiple abscesses in kidneys
11	Fair	C + P	0·092 ,,	42 ,,	Recovery
12	Fair	C	0·106 ,,	56 ,,	Death. Pyelonephritis
18	Poor	C	0·109 ,,	75 ,,	,, Pyelonephritis
23	Very poor	Nil	0·070 ,,	30 ,,	,, Pyelonephritis and malignant prostate
36	Fair	C	0·103 ,,	26 ,,	,, Pyelonephritis
37	Poor	Nil	0·120 ,,	25 ,,	,, Pyelonephritis
46	Fair	P	0·125 ,,	25 ,,	,, No post-mortem. Pulmonary embolism (?)
47	Fair	C + P	0·104 ,,	22 ,,	,, No post-mortem
56	Poor	C	0·118 ,,	48 ,,	,, Pyelonephritis
69	Fair	C + P	0·108 ,,	39 ,,	,, Multiple abscesses in kidneys
73	Poor	Nil	0·105 ,,	24 ,,	,, Pyelonephritis
75	Fair	C	0·083 ,,	28 ,,	,, Pyelonephritis
78	Poor	Nil	0·120 ,,	84 ,,	,, No post-mortem
80	Poor	Nil	0·116 ,,	88 ,,	,, Pyelonephritis and chronic nephritis
83	Fair	P	0·109 ,,	20 ,,	Recovery
86	Poor	Nil	0·122 ,,	56 ,,	Death. Pyelonephritis

C = Cystotomy. P = Prostatectomy. C + P = Two-stage operation. Nil = Catheterization.

ANALYSIS OF DEATHS IN CASES WITH A NORMAL OR HIGH BLOOD-CHOLESTEROL.

Of the 11 deaths occurring in patients with a cholesterol content over 0·130 per cent, an autopsy was obtained in 10 cases (*see Table II*). Only 1 (*Case* 74) showed acute pyelonephritis, and this was a case of adenocarcinoma of the prostate. In addition, *Case* 63 died from a pelvic abscess and fæcal fistula four weeks after removal of the prostate. A false passage had been produced by a metal catheter before the patient's admission to hospital. Free hæmorrhage had resulted, and prostatectomy had been carried out in the single-stage operation. The findings in this case, we think, can scarcely be reckoned on a par with the usual course of a urinary obstruction which terminates in renal sepsis.

The remaining autopsies in the cases with high or normal cholesterol content revealed no evidence of sepsis apart from the usual cystitis, the causes of death being cardiosclerosis (3 cases), acute and subacute nephritis (2 cases), and pneumonia, pulmonary embolism, and post-operative hæmorrhage (1 case each).

Table II.—Analysis of Deaths in Cases with a Blood-cholesterol over 0·130 per cent.

Case No.	Clinical Condition	Operative Treatment	Blood		Cause of Death
			Cholesterol	Urea Nitrogen	
2	Fair	P	0·247 grm.	17 mgrm.	Sudden death—cardiosclerosis
6	Poor	C	0·270 ,,	42 ,,	Malignant prostate and acute nephritis
16	Fair	C	0·354 ,,	31 ,,	Sudden death — cardiac failure
28	Fair	P	0·157 ,,	14 ,,	Pneumonia
44	Fair	P	0·180 ,,	25 ,,	Pulmonary embolism
63	Poor	P	0·167 ,,	22 ,,	Pelvic abscess; fæcal fistula (*see* text)
67	Fair	P	0·257 ,,	87 ,,	Subacute nephritis
72	Fair	P	0·340 ,,	34 ,,	Arteriosclerosis with heart failure
74	Poor	C	0·160 ,,	20 ,,	Malignant prostate and pyelonephritis
79	'Uræmia'	C	0·213 ,,	160 ,,	No post-mortem
85	Fair	P	0·138 ,,	20 ,,	Post-operative hæmorrhage

P = Prostatectomy. C = Cystotomy.

RELATION BETWEEN THE CHOLESTEROL CONTENT OF THE BLOOD AND NITROGEN RETENTION,

From our experience of Folin and Wu's method of estimation of the urea nitrogen of the blood, we consider any figure over 30 mgrm. per cent as significant of defective renal function in middle-aged or elderly subjects. Expressed in terms of urea this is equivalent to values higher than 63 mgrm.

In the present series of 88 patients, 15 gave a blood-urea nitrogen over 40 mgrm. per cent, and therefore suffered from a gross renal inefficiency (*Table III*). It is seen that 8 of these gave at the same time a low blood-cholesterol. In spite of suprapubic drainage to relieve the back-pressure all died except one, post-mortem examination revealing some form of renal sepsis in all 6 cases which were examined.

The remaining 7—that is, those whose blood-cholesterol was not diminished—were drained until the blood-urea content was within the normal range before prostatectomy was carried out, and all recovered except the 2 who showed evidence of the most marked degree of nitrogen retention (*Cases* 67 and 79).

Thus the patients who showed considerable nitrogen retention due to mechanical back-pressure caused by prostatic enlargement, but who reacted well to suprapubic drainage, were those who evidently possessed considerable resistance to bacterial infection, as suggested by a good cholesterol figure. The following are the complete figures of one of our cases :—

Case 49.—Age 76. On admission very ill: uræmic, marked cystitis.

Date	Cholesterol	Urea Nitrogen
Sept. 16, 1922	0·180 grm.	75 mgrm.
Sept. 16, ,,	Cystotomy	
Oct. 2, ,,	0·142 grm.	39·2 mgrm.
Oct. 8, ,,	0·158 ,,	26·2 ,,
Oct. 9, ,,	Prostatectomy.	Convalescence uneventful.

Table III.—Relation between Blood-cholesterol and Nitrogen Retention, 15 Cases with High Blood-urea.

Case No.	Clinical Condition	Operative Treatment	Blood		Results and Post-mortem Findings
			Cholesterol	Urea Nitrogen	
		A.—8 Cases with low blood-cholesterol, of which 7 died.			
10	Fair	C	0·126 grm.	75 mgrm.	Death. Multiple abscesses in kidneys and chronic nephritis
11	Fair	C + P	0·092 ,,	42 ,,	Recovery. Long convalescence
12	Fair	C	0·106 ,,	56 ,,	Death. Pyelonephritis
18	Poor	C	0·109 ,,	75 ,,	,, Pyelonephritis
56	Poor	C	0·118 ,,	48 ,,	,, Pyelonephritis
78	Poor	Nil	0·120 ,,	84 ,,	,, No post-mortem
80	Poor	Nil	0·116 ,,	88 ,,	,, Pyelonephritis and chronic nephritis
86	Poor	Nil	0·122 ,,	56 ,,	,, Pyelonephritis
		B.—7 Cases with normal or high blood-cholesterol, of which 2 died.			
13	Fair	C + P	0·173 ,,	40 ,,	Recovery
25	Fair	C + P	0·206 ,,	62 ,,	,,
49	Fair	C + P	0·180 ,,	75 ,,	,,
50	Fair	C + P	0·164 ,,	56 ,,	,,
64	Fair	C + P	0·143 ,,	42 ,,	,,
67	Fair	C + P	0·257 ,,	87 ,,	Death. Subacute nephritis; cholelithiasis
79	Fair	C	0·213 ,,	160 ,,	,, No post-mortem

C = Cystotomy. C + P = Two-stage operation. Nil = Catheterization.

On the other hand, the results were almost uniformly unsatisfactory in cases showing considerable nitrogen retention with, in addition, a low cholesterol content.

There was no fall in blood-urea nitrogen after cystotomy—indeed the nitrogen retention was often more marked—after a fortnight's drainage. An example of such a finding is the following :—

Case 12.—Age 73. Clinically 'fair condition'. No pyrexia.

Date	Cholesterol	Urea Nitrogen
Aug. 10, 1922	0·106 per cent	56·6 mgrm. per cent
Aug. 10, ,,	Cystotomy	
Aug. 21, ,,	0·100 per cent	72·8 mgrm. per cent
Sept. 13, ,,	Death. Acute pyelonephritis with chronic cystitis.	

Such findings suggest extension of sepsis to the portion of renal tissue still functioning, while they afford evidence that "the occurrence of sepsis at the time of, or following operation, may damage the remaining portion of renal substance, and therefore put the patient in grave danger."[10]

THE POST-OPERATIVE FALL IN BLOOD-CHOLESTEROL.

The above findings raise the question: "Does suprapubic drainage ever raise the cholesterol content as well as lower the blood-urea?"

Such an increase in the cholesterol value is rarely to be seen, and herein lies the chief fallacy to be guarded against in assessing the significance of cholesterol estimations. Other factors than those associated with antibody formation appear to influence the blood-cholesterol, and special emphasis must be laid on the fall in the lipoid content of the blood, which appears to follow any operative procedure. This was strikingly shown in the course of an investigation carried out by us[7] on the effects of splenectomy on cholesterol metabolism in acholuric jaundice and splenic anæmia. After removal of the spleen there was invariably a fall in blood-cholesterol during the first fortnight, which was uniformly low in the conditions investigated, to be followed by a rapid and progressive increase in lipoid content during the next few months.

A similar post-operative fall was a common observation in many of the cases of the present investigation. Even a minor operation, such as a suprapubic cystotomy, whether performed under a local or general anæsthetic, produces this effect. The flaring up of sepsis would doubtless be a contributing factor, but this will not explain the fall in many of the cases after operation.

BLOOD-CHOLESTEROL IN MALIGNANT DISEASE OF THE PROSTATE.

Luden[11], working at the Mayo Clinic, has shown that the blood-cholesterol is usually high in malignant disease. In our series of 88 cases, there were 14 suffering from carcinoma of the prostate, and in these the average blood-cholesterol was 0·178 per cent. Only one gave a distinctly low value, viz., *Case* 23, in which the cholesterol was 0·070 per cent, one of the lowest values of the series, while there were two borderland cases with 0·128 per cent. The patient (*Case* 23) was distinctly febrile at the time of investigation, his general condition was very poor, and he suffered from a severe and foul cystitis. Post-mortem examination revealed multiple abscess formation in the kidneys.

It is difficult to determine the number of these cases that were suffering from renal sepsis, as most of the patients were discharged from hospital as soon as the diagnosis of malignancy was established and their urgent symptoms were relieved. Two others of these 14 patients died while in hospital, viz., *Case* 74, with a cholesterol content of 0·160 per cent, and in whose case pyelonephritis was found at autopsy, and *Case* 6 who died suddenly, and whose blood-cholesterol was 0·270 per cent. Autopsy revealed a hæmorrhagic cystitis with a recent acute nephritis, but there was no septic infection of the kidneys or other organs. The adrenals were distinctly rich in cholesterol, while it may be noted in connection with the hypercholesterolæmia that the patient also suffered from cholelithiasis.

It appears, therefore, that the blood-cholesterol in malignant disease of the prostate is of a higher average value than that of patients suffering from simple adenomatous enlargements. Hence a case of prostatic carcinoma may give a value considerably above 0·126 per cent although the patient may be suffering from pyelonephritis, a low figure being obtained only in cases of advanced and extensive infection. This exception, however, does not detract from the value of cholesterol estimations in the benign form of prostatic enlargement, since the preliminary cystotomy or other mode of examination is usually sufficient for the diagnosis of carcinoma.

SUMMARY WITH CONCLUSIONS.

1. In the treatment of cases of urinary obstruction due to prostatic enlargement, two factors have to be taken into account in deciding on what operative procedure shall be required, and when, viz.: (*a*) The degree of defective renal function present; (*b*) The capacity of the patient to resist the spread of sepsis to the upper urinary tract, since a cystitis of greater or less degree is usually present.

2. An endeavour has been made to discover a test which should be of practical value in estimating this factor of sepsis, especially in cases where the clinical signs of its presence are few or uncertain. We find that the cholesterol content of the blood appears to be a fair measure of the degree of resistance to infection possessed by the individual, and are of the opinion that such estimations form a useful practical guide to the degree of operative risk in cases of urinary obstruction.

3. Of unselected cases of prostatic obstruction, 88 have been investigated, of which 18 gave a low cholesterol value, viz., less than 0·130 per cent. Of these, 16 died of pyelonephritis, while 2 recovered. Of the other 11 deaths in the series in which the blood-cholesterol was 0·130 per cent or higher, only one death was due to an ascending urinary infection.

4. In only about 50 per cent of the patients with a low cholesterol value who subsequently died of pyelonephritis did clinical opinion of the general condition of the patient contra-indicate operation. Moreover, all these cases showed more or less defect in renal function, upon which, rather than upon the latent sepsis, did the clinical manifestations depend.

5. In many instances, therefore, no certain information as to the resistance of the patient to post-operative spread of sepsis is afforded by considering the patient's clinical condition alone. Nor does the degree and type of local sepsis in the form of the cystitis, which is present in greater or less degree in all such cases, provide reliable information as to the powers of resistance of the individual. In cases of marked cystitis, both high and low cholesterol values were obtained.

6. We suggest that a low 'blood-cholesterol' in a case of acute or chronic retention due to prostatic enlargement is significant of a low capacity for antibody formation, and points to the case being a bad operative risk as regards prostatectomy. The value of cholesterol estimations is further enhanced by their affording information as to a low bacterial resistance, even when the clinical manifestations of such are absent or uncertain.

7. Genito-urinary sepsis with the risk of ascending infection is of more serious prognostic import than nitrogen retention from defective renal function. Thus a high blood-urea alone is not necessarily serious as to ultimate prognosis, since cases of nitrogen retention which gave a normal or high cholesterol value acquired a normal blood-urea content after a period of suprapubic drainage. When prostatectomy was thus postponed, uniformly successful results were obtained, and a rapid convalescence ensued.

On the other hand, the combination of a high blood-urea and a low cholesterol content is of very serious prognosis, and points to an undoubtedly bad surgical risk. Of 8 such cases in this series, all died save one.

8. The test described is of much less value in cases of malignant disease of the prostate, where high or normal cholesterol figures are often present even when pyelonephritis exists. Low figures are obtained only in advanced or extensive infection. This discrepancy is probably to be accounted for by the high cholesterol content of the blood commonly met with in malignant disease.

To the surgeons of the Leeds General Infirmary we desire to express our thanks for their courtesy in permitting us to carry out these observations on cases under their care. We would especially acknowledge our indebtedness to Mr. J. F. Dobson, at whose suggestion we took up this investigation, for his constant valuable criticism and advice, and to Professor M. J. Stewart for affording us facilities for carrying out the laboratory work in the Pathological Department of the University of Leeds.

REFERENCES.

[1] *Proc. Roy. Soc. Med.* (Urol. Sect.), 1922, xv, 7 and 55.
[2] DOBSON, *Brit. Med. Jour.*, 1921, i, 289.
[3] WALKER, *Proc. Roy. Soc. Med.* (Urol. Sect.), 1922, xv, 64.
[4] WHITNEY, *Arch. Internal Med.*, 1917, xx, 931.
[5] LANDAU, *Deut. med. Woch.*, 1913, xxxix, 546.
[6] BARBARY, *Bull. de l'Acad. de Méd.*, 1916, xxvi, 221.
[7] MACADAM and SHISKIN, *Quart. Jour. Med.*, 1923, xvi, 193.
[8] FOLIN and WU, *Jour. of Biol. Chem.*, 1919, xxxviii, 83.
[9] CHAUFFARD LAROCHE, and GRIGAUT, *Obstétrique*, 1911, iv, 481.
[10] *Proc. Roy. Soc. Med.* (Urol. Sect.), 1922, xv, 67.
[11] LUDEN, *Jour. of Lab. and Clin. Med.*, 1918, iv, 849.

JUXTA-AMPULLARY CARCINOMA OF WIRSUNG'S DUCT.

By GEOFFREY HADFIELD, Bristol.

INTRODUCTION.

Cancer arising in the ampulla of Vater or in the juxta-ampullary portions of the ducts which open into it has considerable surgical interest. The growths themselves are comparatively miniature in size and relatively benign; severe obstructive symptoms appear promptly, often before metastases are found; and except that jaundice is usually severe enough to add considerably to the operative risks, these cases are often favourable to surgical interference.

The anatomical localization of these tumours is often difficult: their histological characters seem to afford the safest criteria of their origin. Rolleston has collected 19 cases of cancer arising in the plicated gland-bearing mucosa of the ampulla itself, and mentions that of 21 cases recorded as being "cancer of the ampulla", only 6 appear to be true cancer "du pylore pancréatico-duodénale" of Hanot. Some of these growths arise in the termination of the common bile-duct and are columnar-celled, whilst carcinoma of the mucous membrane covering the duodenal surface of the papilla, or arising in its contained Brunner's glands, would seem to be relatively frequent. Juxta-ampullary carcinoma of the pancreatic duct has been seldom described. Carnot and Harvier[1] and Luzzato[2] have published undoubted cases. The following case is of interest as being an example of juxta-ampullary cancer of this type, and, in addition, presents several points of interest in diagnosis.

CASE REPORT.

T. G., male, age 48, was admitted to the General Hospital, Bristol, on Aug. 3, 1923, complaining of jaundice and increasing weakness.

History of Present Illness.—Was in good health until the end of June, 1923, five weeks before admission. Then had an attack of 'gastritis' with severe epigastric pain and vomiting, was away from work one week, noticed general irritation of skin. About the middle of July the vomiting recurred, he noticed he was often giddy, his appetite was poor, and there was increasing weakness. Jaundice appeared at the end of July; the weakness, anorexia, and vomiting continued. He noticed that he was losing weight, that his stool was clay-coloured, and that any fatty food made him vomit.

Past History.—Twenty years ago a vague of history of "catarrh of the liver", but no jaundice. No other illness. Venereal infection denied. Never a heavy drinker.

Condition on Admission.—A thin, spare man, rather deeply but not intensely jaundiced; the tint of the skin and sclerotics was a rather deep yellow. Liver dullness extended nearly half-way to the umbilicus in the

middle line. Surface smooth; gall-bladder not felt. No abnormality in chest. Nothing felt per rectum. Urine deep yellow in colour, loaded with bile pigment; no excess of urobilin present.

Blood.—Wassermann reaction, negative. Van den Bergh reaction, delayed direct type: 17·5 units.

Fæces.—Total fat 30 per cent of dried weight; neutral fat 17 per cent of dried weight; fatty acid 13 per cent of dried weight; i.e., slight excess, but fairly well split. (N.B.—This figure is probably explained by the fact that (1) obstruction to the common bile-duct was partial, and (2) an accessory pancreatic duct was present.)

Course of Illness.—The weakness increased: he was often drowsy, and occasionally irritable and irrational. The jaundice steadily deepened; a few petechiæ appeared in the skin. There was no fever. Anti-syphilitic treatment had no effect on the signs or symptoms. A diagnosis of extra-mural obstruction of the common bile-duct, probably malignant in nature, and located in the head of the pancreas, was made, and exploratory laparotomy decided upon. The liver was found enlarged and smooth. The gall-bladder, almost completely concealed by the enlarged liver, was the size of a large William pear. No thickening of its wall, adhesions, or gall-stones were found. The common bile-duct was considerably dilated, and along its course in the head of the pancreas a chain of four or five closely-set, enlarged glands could be felt. No stones were palpated in the extra-hepatic ducts; the head of the pancreas felt rather swollen and somewhat nodular. A condition of non-calculous obstruction of the common duct in the region of the head of the pancreas was diagnosed, and an anastomosis between the gall-bladder and the antero-superior surface of the stomach performed, this being considered the most suitable operation for cases of this type. The gall-bladder on aspiration was found to contain a thin, opalescent, and practically colourless fluid. Little shock followed the operation, and for three days progress was all that could be desired. On the fourth day collapse symptoms appeared, there was a little vomiting, and the patient died thirty-six hours after the onset of these symptoms.

POST-MORTEM EXAMINATION (*Fig.* 283).

On palpation of the pancreas, several hard fixed nodules were felt in the posterior part of the head and between it and the duodenum. On opening the latter, the biliary papilla was found considerably enlarged and very prominent, projecting about 1 cm. from the surface of the intact mucous membrane of the bowel.

The tip of a No. 6 rubber catheter passed easily into the papilla for a distance of a little less than 1 cm.; by pressing on the head of the pancreas a little thick brown bile could be expressed from the orifice; a probe encountered obstruction just inside the ampulla, but could be passed with some difficulty for several inches into the common bile-duct. On opening up the papilla, a well-formed ampulla was exposed, projecting into which was a spherical mass of new growth, 1·5 cm. in diameter, arising by a broad base of attachment from the posterior wall of the termination of the pancreatic duct. The growth half filled the ampulla, which appeared dilated; its mucous membrane was not

infiltrated. There was no infiltration of the wall of the common duct, which was almost completely occluded, and deviated to one side by pressure of the growth. The growth was white, hard, and sessile, and its surface slightly roughened. The duct of Wirsung, the common bile-duct, and the hepatic ducts were considerably dilated; the cystic duct appeared rather narrower than normal. Thick, dark, deep-brown fluid was present in the common bile-duct. No calculi were found.

An accessory pancreatic duct was found opening separately into the duodenum. Partly embedded in the posterior portion of the head of the pancreas, and between it and the second part of the duodenum, were four enlarged fixed lymphatic glands, the largest 1·5 cm. in diameter.

The gall-bladder was enlarged (long axis 15 cm.), its walls were slightly thickened and rather rigid; the mucous membrane, after washing off some loose clot, was white, smooth, and rather shiny. The cavity was filled but not distended by recent clot from hæmorrhage about the operation site. The anastomosis was firm and watertight. The liver was olive-green in colour, slightly shrunken, and very flabby. The surface was smooth. The intrahepatic ducts were considerably dilated and contained thick brownish-yellow bile. The portal canals were prominent and the lobular pattern very distinct, each lobule showing as a deep green spot fading to yellow-brown at its periphery. The structure was homogeneous throughout; the organ appeared anæmic; no suggestion of gross hepatic necrosis. The spleen was small and shrunken, the capsule wrinkled, and the pulp pale and firm. The pancreas showed no obvious naked-eye abnormality except for dilatation of its main duct.

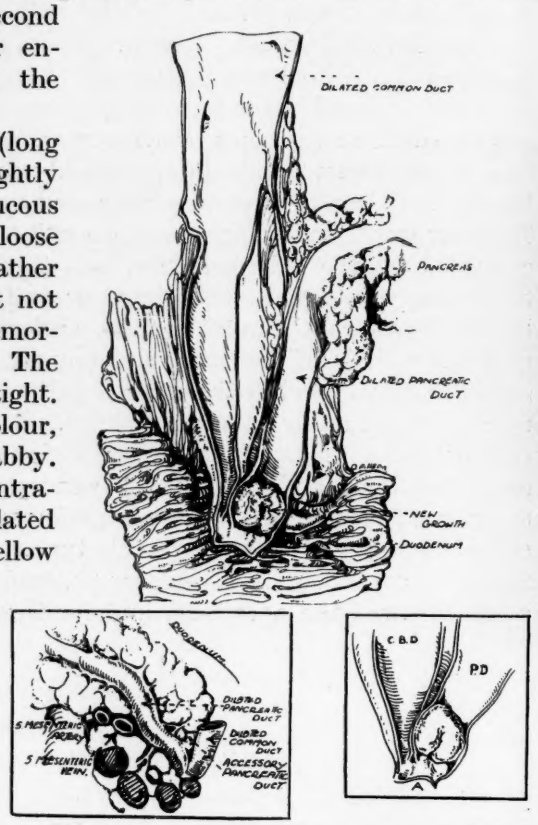


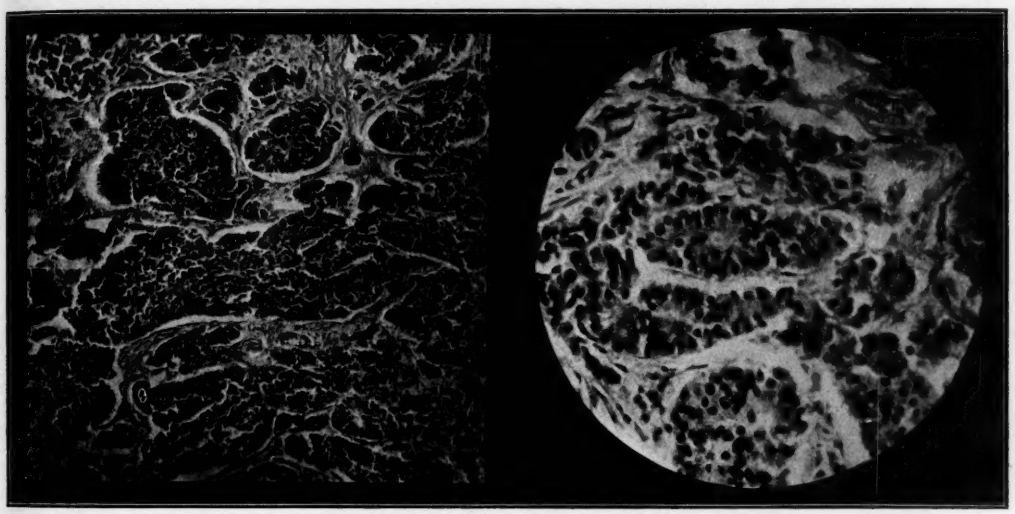
FIG. 283.—Carcinoma of the ampulla of Vater originating in the termination of the pancreatic duct. The left lower figure shows the pancreas from behind, with position of enlarged glands.

PATHOLOGICAL HISTOLOGY.—*Growth.*—Sections show large isolated masses of undifferentiated spheroidal-celled carcinoma lying in rather dense fibrotic stroma. There is no tendency to tubule formation; the cells are large, spherical, and in close opposition. There is a slight superficial ulceration (microscopic). The cell type is very constant; no columnar or goblet cells seen.

Gland.—No trace of lymphatic tissue present. Normal tissue is replaced by massive solid and anastomosing alveoli composed of spherical cells of the

same type as in the primary growth (*Fig.* 284). Where the stroma is more abundant there is considerable lymphatic permeation. Unlike the primary growth, there is occasional tubule formation in the metastases.

Pancreas.—Sections show considerable dilatation of the larger ducts, with flattening, and in some places localized hyperplasia and papillary projection, of the lining epithelium. There is a general interlobular pancreatitis of moderate intensity, most marked about the larger ducts. In those lobules bordering on the main connective-tissue septa small groups of tubules are isolated by fibrosis, but this has not penetrated into the more deeply placed lobules, the acini and islets of which show no special change. The gland shows no atrophic changes, and the appearances seen are apparently secondary to obstruction of the duct system only.



A B

FIG. 284.—Retropancreatic gland (juxta-ampullary carcinoma of Wirsung's duct). A, Low-power view showing massive columns of spheroidal cells which entirely replaced the lymphatic tissue of the gland. B, High-power view, showing undifferentiated spheroidal-celled carcinoma.
The primary growth was of the same type.

Liver.—The larger bile-ducts are much dilated and their epithelium is flattened and desquamating. A moderately intense inflammatory cell-exudate of lymphocytes and a few plasma cells surrounds them. The medium-sized ducts are proportionately less dilated, but their portal canals contain more cells, and in addition many nuclear fragments, while fibroblasts are scanty. The dilatation of the ducts in the smallest portal canals is slight and their epithelium little altered, but many of these ducts contain plugs of dark green or yellow-brown bile. Almost every liver lobule shows an area disposed around the central vein where bile stasis and necrosis of the hepatic parenchyma are obvious. This area usually occupies the inner third of the lobule and in some cases stretches across it to join similar areas in adjacent lobules. The outer two-thirds of the lobules are often almost normal-looking except that the bile capillaries are full. In the degenerate central areas the liver-cell

columns are broken and the cells isolated and filled by pigment. Many of these cells have pyknotic nuclei; in some the nucleus is fragmented or absent and the cell body only faintly outlined, whilst masses of pigment and nuclear fragments occupy the place of others.

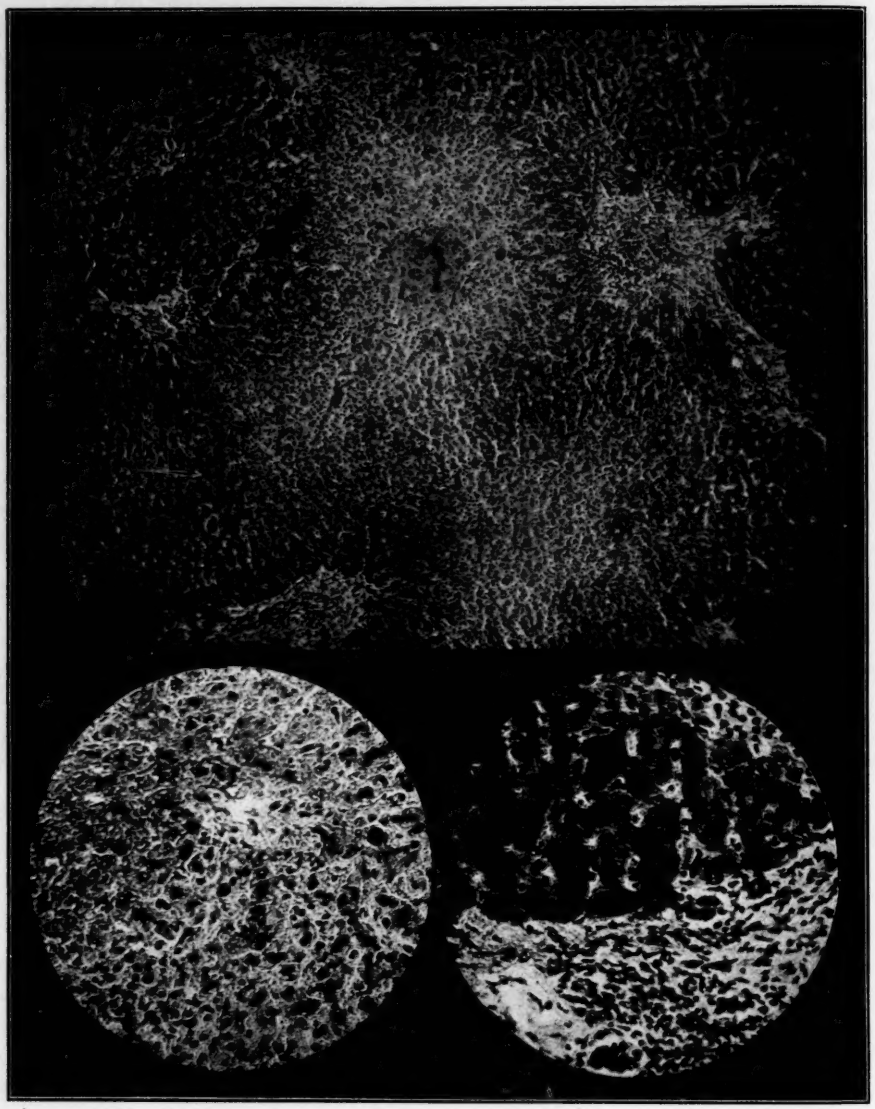


FIG. 285.—Liver from case of juxta-ampullary carcinoma of pancreatic duct, showing atrophy from extra-hepatic biliary obstruction. A, Low-power view: lobules show centri-lobular pressure atrophy of parenchyma. B, Atrophic parenchyma from centre of a lobule. C, Fairly well-preserved cells of edge of lobule.

Large masses of bile-pigment lie free in the central area of the majority of the lobules. In some lobules there are many centrally-placed phagocytic cells with large, clear, well-staining nuclei, containing pigment in their cytoplasm. The liver-cell necrosis is seen in the neighbourhood of the central vein of the

majority of the lobules and is present in all sections. The histological changes (*Fig*, 285) are those often found in association with complete aseptic obstruction of the common bile-duct. The total amount of necrosis of liver parenchyma is considerable and regeneration absent.

SUMMARY.

A previously healthy man of 48 died after an illness of three and a half months, characterized by deepening jaundice, without definite intermission, and increasing weakness.

Post-mortem examination showed a sessile nodule of carcinoma the size of a pea arising in the wall of Wirsung's duct just above the dilated ampulla of Vater, and obstructing the common bile-duct by lateral pressure. Metastases were present in the retropancreatic glands. The nodule of growth was the average size and shape of the enlarged glands, and situated so near them that it was extremely difficult to distinguish between the growth and the enlarged glands by palpation of the duodenum at the operation. The gall-bladder was found distended by clear colourless fluid at operation.

A functioning accessory pancreatic duct was present. The Van den Bergh reaction and a stool analysis did not materially assist the clinical diagnosis. Death was probably due to widespread pressure atrophy of the liver cells, secondary to obstruction of the common duct.

The case reported presents the following points of interest, apart from the rare anatomical localization of the tumour :—

1. Absence of definite intermissions in the intensity of the jaundice.
2. Concealment of the enlarged gall-bladder by the swollen liver—an apparent exception on clinical examination to Courvoisier's law.
3. The retropancreatic glandular metastases were of the same size and consistence as the miniature growth itself : this was therefore mistaken for an enlarged gland, and the duodenum was not opened.
4. There was complete sequestration of the gall-bladder which contained colourless watery fluid : the grave significance of this is borne out by the extensive atrophy of the liver parenchyma found post mortem.
5. The relatively benign nature of the tumour is striking : there was no infiltration of the mucous membrane of the bowel, and the lymphatic metastases were limited to the retropancreatic set of glands.

I am indebted to Mr. Clifford Moore, Surgeon to the General Hospital, Bristol, for permission to use his clinical records.

REFERENCES.

[1] CARNOT and HARVIER, *Bull. et Mém. Soc. méd. Hôp. de Paris*, 1906, 3rd series, xxiii, 296.

[2] LUZZATO, *Clin. Med. Ital.* Milano, 1902, xli, 282.

A METHOD OF TREATMENT OF FRACTURE OF A SINGLE CONDYLE OF THE FEMUR WITH DISPLACEMENT BACKWARDS OF THE DISTAL FRAGMENT TO THE POPLITEAL SPACE.

By LIONEL SUTCLIFFE, Chesterfield.

The first surgeon to describe a fracture of a single condyle of the femur was Bichat in 1850, but he confined himself to pointing out its possibility. Malgaigne's[1] would appear to be the earliest description of a series of cases, and he, in 1859, confined himself to three only, which he had seen and treated personally. It is interesting to note that he suggested—for cases with little or no displacement—the best treatment to be rest in the extended position of the joint over a cushion after reduction. The application of leeches and evaporating lotion to reduce the swelling followed, and finally he surrounded the joint with a trough of strong pasteboard, previously softened in warm water, in order to conform to the contours of the parts.

The rarity of this condition, together with the considerable barrenness of authoritative information, either with regard to such an injury or to its treatment, has led me to believe that the following remarks may suggest a method of procedure of value in such cases. Most text-books dismiss a fracture in this region airily, by stating that they are all very rare, but that small chips of the condyle may be separated from the femur in certain cases of injury to the knee-joint.

On Feb. 11, 1924, T. E., a male, age 20, a collier, was admitted to the Chesterfield Royal Hospital under my care, with an injury to the right knee-joint. Whilst playing football on Feb. 9, the patient caught his right foot between another player's legs, and fell heavily to the ground, twisting his leg as he did so. He had been unable to put any weight on the limb since, and all movements of the joint were painful. Examination disclosed a considerable amount of swelling around the whole joint, which was mainly extra-capsular. No floating of the patella could be detected, and both patella pouches showed fullness. The position of the joint was one of complete extension together with eversion of the leg. Limitation of flexion was absolute. Crepitus was elicited by gentle friction of the condyles. Increased lateral movement of the tibia and fibula with the femur was present. There was a fullness in the popliteal space, most marked on the inner side. The X rays showed a fracture of the medial condyle of the femur, with complete avulsion, and displacement backwards to the popliteal space.

Such were the history, clinical signs, and X-ray report two days subsequent to his injury. The more natural condition to follow such an injury in a patient of this age is the separation of the lower epiphysis or a T-shaped fracture of

both condyles. It is rather remarkable that, although this patient is 20 years old, the femur elected to fracture higher up, rather than follow the more natural course of separating at the epiphyseal junction.

Roberts and Kelly[2] suggest that forced abduction or adduction of the knee, twisting of the leg, or direct force transmitted through the head of the tibia toward the involved condyle may cause this fracture. From a study of the history of this case, it appears clear that a combination of all these

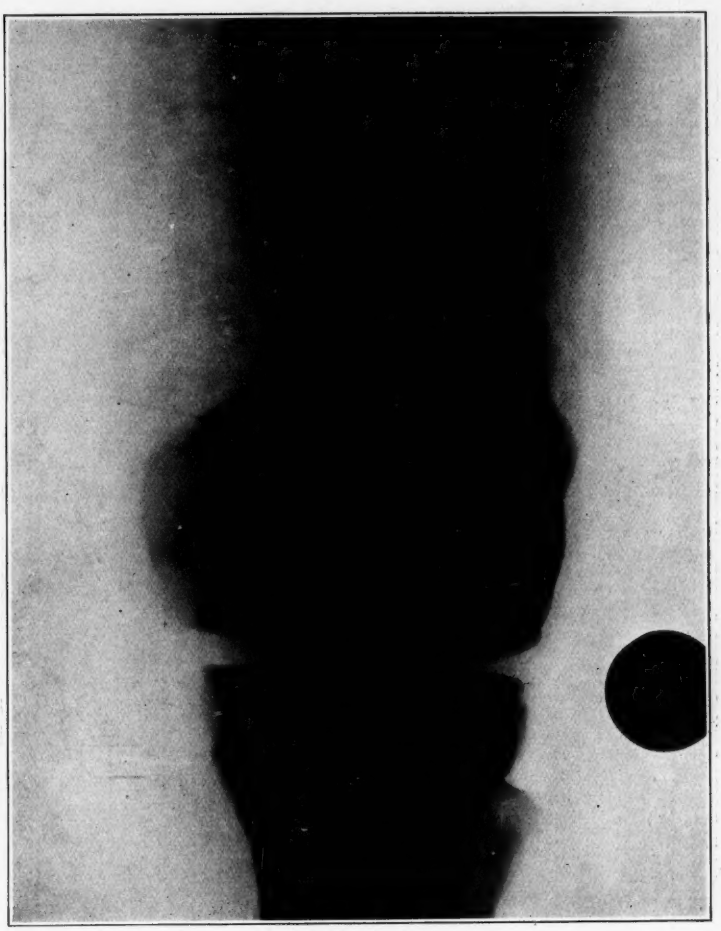


FIG. 286.—The fracture and displacement before operation.

factors was the etiological feature in fracturing the condyle. Abduction and twisting, at the moment the patient's leg was between the other player's legs, and direct violence when he was forcibly thrown to the ground.

Although no precedent could be found, it was decided that conservative treatment by manipulation and splinting was contra-indicated, (1) owing to the amount of effusion, with the consequent impossibility of accurate reduction, and (2) owing to fear of damage to the popliteal vessels from the forcible reduction of the backward displacement of so large a fragment.

Therefore on Feb. 14, 1924, a longitudinal incision, 4 in. long, was made, under general anæsthesia, on the medial aspect of the patella, which was deepened down to the vastus medialis. There was a distinct rent in this muscle, which was cleared of blood-clot, and increased upwards and downwards for the length of the incision. The lower end of the femur, external to the capsule, was exposed through the vasti lateralis and intermedius. It was apparent here that the tendinous expansions from the vasti lateralis and

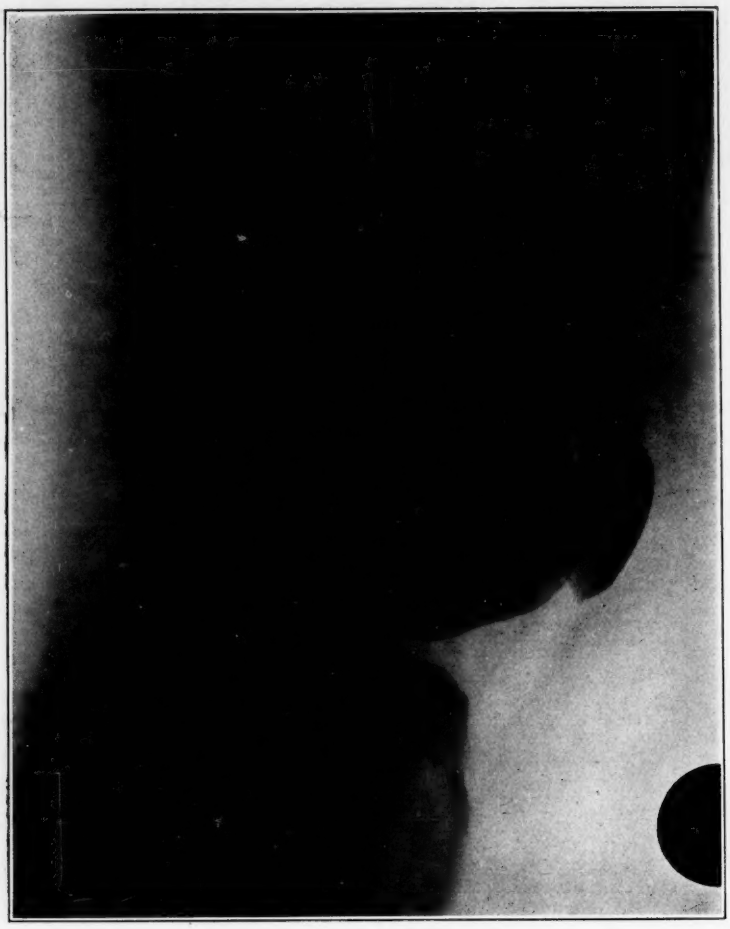


FIG. 287.—The fracture and displacement before operation.

medius, which should strengthen this portion of the capsule, were very poorly developed. The capsule was incised at right angles to the skin incision, clots and serum were sponged out, and the proximal fragment was sought for and isolated. The condyle was then discovered completely separated from the femur, turned round on itself from without inwards, and displaced upwards and backwards to the popliteal space. The line of fracture ran obliquely downwards and inwards from the femoral shaft to the intercondylar space, and was entirely intracapsular.

There appeared so little likelihood of a satisfactory union resulting if left in position after reduction that it was decided to plate the fragments. A small Lane's plate with three screws was used for this purpose, one screw driven home below the line of fracture on to the condyle, and two screws above the line of fracture on to the femur. After careful cleansing of the joint by dry swabbing and complete hæmostasis, the capsule was closed by interrupted catgut sutures, the rent and incision in the muscle being closed similarly, without drainage, and after skin suture the joint was put up on a double inclined plane, at an

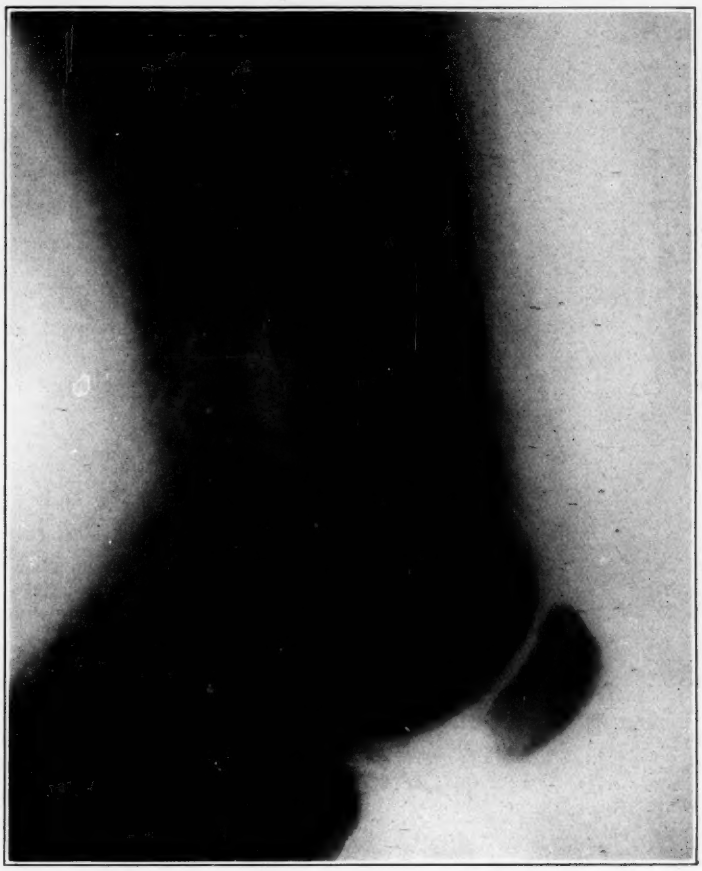


FIG. 288.—Condition of the fragments after plating.

angle of 45°. After fourteen days the skin sutures were removed, and the wound had healed by first intention.

Gentle massage for twenty minutes daily was commenced on the fifteenth day, and carried on for one week. Passive movement was added at the end of the first week's massage, and then active and passive movement instituted two weeks after removal of the skin sutures.

The patient remained in hospital for eleven weeks, and was discharged cured, with a normally functioning knee-joint, but with instructions not to place full weight on the knee for another four weeks. He can now walk

normally without aid, complete range of movement is present in the joint, and there is no undue lateral movement.

The series of X-ray photographs (*Figs.* 286–289) explain themselves.

Stimson[3] affirms all fractures of a single condyle to be rare, and does not describe any case with complete avulsion. He says that the displacement is slight and—most important—the connections are untorn. Non-union of the fragment and suppuration of the joint, with or without a fatal termination, may result, in his experience, from conservative non-operative treatment.

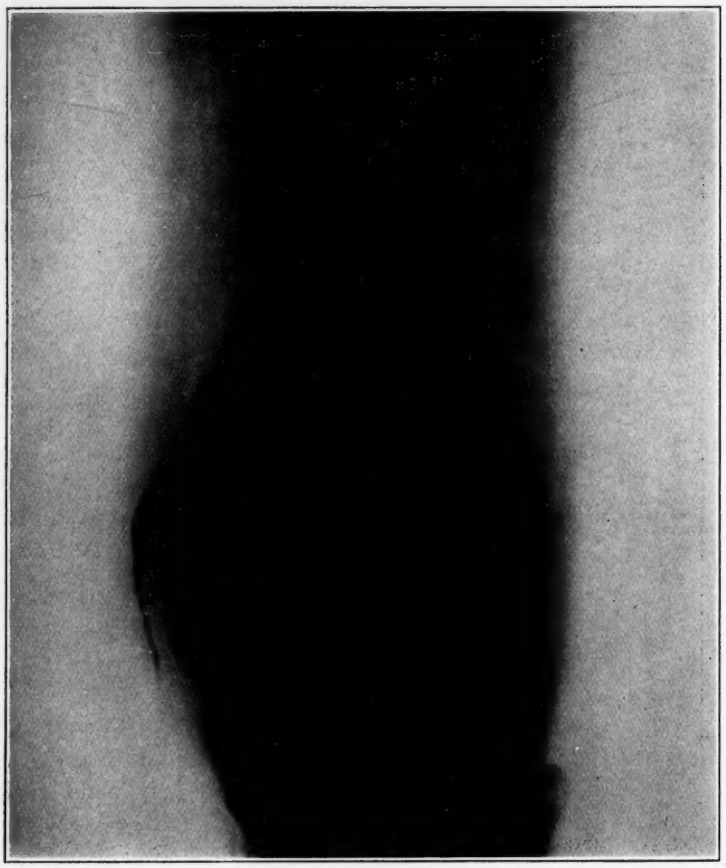


FIG. 289.—Condition of the fragments after plating.

He recommends reduction and immobilization, with the knee fully extended, owing to the fact that the lateral ligament is usually intact, and will in the extended position be tense, and thus aid fixation.

A. H. Crosby[4] reports a case in which the ununited fragment was removed six months after injury, and the patient made a complete recovery.

Roberts and Kelly refer to manipulative treatment of the fragments and immobilization in splints, but suggest no operative procedure. I am confident that in this case imperfect reduction owing to the laceration of the lateral ligament, danger to the popliteal vessels, and an impaired function of the joint,

would almost certainly have resulted from manipulation without fixation by open operation.

CONCLUSIONS.

1. An intracapsular fracture of a condyle of the femur can be plated.
2. The method of approach to the joint was justified by the traumatism caused by the accident.
3. The risk of an open operation was justified by the age of the patient and the importance to him of a sound knee, because of his occupation as a miner.

My sincere thanks are due to Dr. Morrell for his help in preparing the X-ray films, and to Dr. Webster for his unremitting care and assistance in the after-treatment.

REFERENCES.

[1] MALGAIGNE, *Treatise on Fractures*, 592.
[2] ROBERTS and KELLY, *Fractures*, 575.
[3] STIMSON, *A Treatise on Fractures and Dislocations*, 404.
[4] CROSBY, A. H., *New Hampshire Jour. of Med.*, 1857.

ON RETROPERITONEAL HERNIA: WITH A REPORT ON THE LITERATURE.

By A. RENDLE SHORT, Bristol.

In 1906 a monograph was published by Moynihan classifying the various types of retroperitoneal fossæ in which a hernia has been found, and giving the literature up to that date. In 1915 the present writer took advantage of a case occurring in his practice to report on the recorded cases from 1906 to that time. Another patient suffering from this rare and interesting condition has lately been under his care, and as a further period of nine years has elapsed and some new types of hernia have been described, it seems a suitable opportunity to bring the literature up to date and to summarize it. He will not on this occasion describe cases which have been unsuspected during life and discovered post mortem, but confine himself to those in which there were symptoms and, usually, an operation.

Case.—C. B., male, age 20, admitted to the Bristol Royal Infirmary in August, 1923, suffering from intestinal obstruction. The history given was that he had been subject to numerous attacks of the same type all his life, having had forty or fifty in all. They usually lasted about two or three days, and then passed off. In each attack there was abdominal pain, vomiting, stoppage of the bowels, and some distention. Nothing special was to be made out on examination of the abdomen, except some general distention of the small-gut type. I waited a day or two to see if it would pass off, but as it did not, I operated on Aug. 18, and found that many feet of jejunum were herniated into a typical left paraduodenal fossa of Landzert, showing the inferior mesenteric vein in the free border of the sac. The coils of gut were withdrawn, but as the condition was bad, and the gut very distended, no attempt was made at that time to close the mouth of the sac. There was some suppuration of the abdominal wall, but the patient recovered, and had no further attacks. On Sept. 29, 1923, I operated again to close the sac, but found that the mild adhesive type of peritonitis which accompanied the suppuration of the abdominal wall had made a recurrence impossible by adhesions of the jejunum and plastic closure of the mouth of the sac.

Brief Description of the Retroperitoneal Fossæ.—The frequency of occurrence of the principal fossæ is given as follows by Desjardins:—

Inferior duodenal fossa of Treitz ..	60 per cent
Combined inferior and superior ditto (*Fig.* 290)	30 ,,
Superior duodenal alone	5 ,,
Left paraduodenal fossa of Landzert ..	2 ,,
Mesocolic fossa	2 ,,
Right mesentericoparietal fossa of Waldeyer	1 ,,

Of these, the superior and inferior duodenal fossæ do not appear to be of pathological importance.

The paraduodenal fossa of Landzert, or left duodenojejunal fossa, lies to

the left side of the fourth part of the duodenum, the opening looks to the right, and the inferior mesenteric vein runs in the free border (*Fig.* 291).

The mesentericoparietal fossa of Waldeyer, or right duodenojejunal fossa, lies in the first part of the mesentery of the jejunum, below the duodenum.

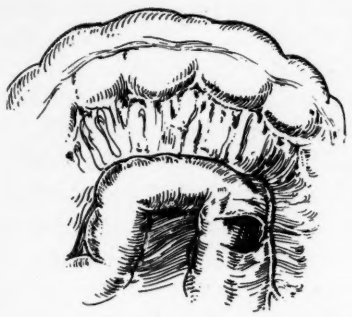


FIG. 290.—Combined superior and inferior duodenal fossæ.

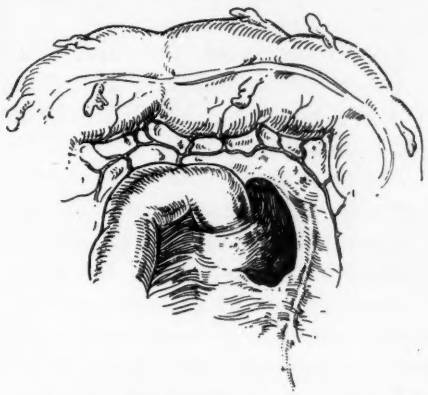


FIG. 291.—Paraduodenal fossa of Landzert, with inferior mesenteric vein in neck of sac.

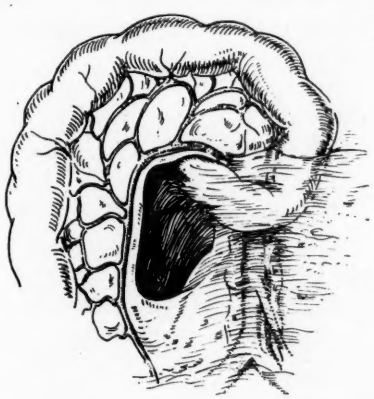


FIG. 292.—Mesentericoparietal fossa of Waldeyer. Shows the superior mesenteric artery in the neck of the sac.

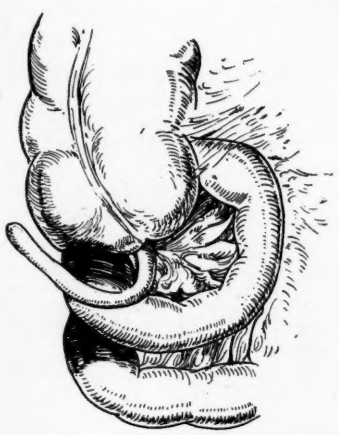


FIG. 293.—Retrocolic fossa (Pribram's case).

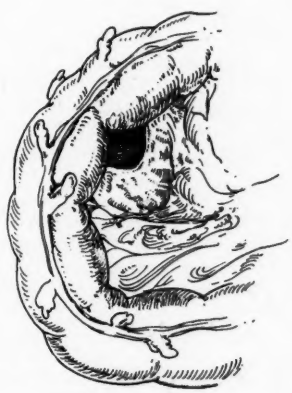


FIG. 294.—Intersigmoid fossa (Macholl's case).

The opening is to the left, and the superior mesenteric artery runs in the free edge (*Fig.* 292).

The retrocolic fossa lies behind the cæcum and ascending colon, with the mouth looking downwards. *Fig.* 293, illustrating this fossa, is redrawn from Pribram's case.

The ileo-appendicular fossa lies between the abnormal ileo-appendicular fold (so-called bloodless fold of some authors) and the mesentery of the appendix; the termination of the ileum lies above it. The opening looks to the left and downwards.

The fossa ileocæcalis of Hartmann lies between the mesoappendix and the attachment of the mesentery to the iliac fossa running from the ileocolic angle to the posterior and inner aspect of the cæcum. The fossa looks downwards.

The intersigmoid fossa is situated in the mesosigmoid, on the left-hand side, close to the attachment of the mesosigmoid to the posterior wall. The opening looks downwards. It is present in the majority of children. But in some cases it lies within the layers of the mesosigmoid, as in the example shown in (*Fig.* 294), re-drawn from Macholl's case, where the fossa lies much nearer to the bowel than to the parietes.

Andrews, in a recently published paper, argues that so-called retroperitoneal hernia is really due to a developmental abnormality, and not to an acquired condition.

Table I.—Cases of Retroperitoneal Hernia Producing Symptoms Recorded since 1914.

Date	Reporter	Sex and Age	Description	Treatment	Result
Hernia of Left Paraduodenal Fossa.					
1914	Davis	M 48	Symptoms of strangulation, globular swelling felt. One previous attack	Reduction	Died
1916	Pringle	M 40	Recurrent attacks of obstruction. Tumour felt	Partial reduction; short-circuiting	Cured
1916	Wagner	F 14	Recurrent attacks; obstruction	Reduction	Cured
1917	Bramlett and Ashhurst	M 47	Recurrent pain; acute obstruction	Reduction	Died
1917	Filauro	F 60	Acute obstruction; cyst-like swelling	Reduction	Died
1917	Nixon	M 30	Recurrent abdominal pain	Reduction	Cured
1918	Grad	F 52	Abdominal pain	Reduction	Cured
1920	Haberer	F 62	Acute obstruction	Reduction	Cured
1920	Harttung				
	1.	M 49	Acute obstruction	Reduction	Cured
	2.	M 44	Acute obstruction	Big resection of bowel	Cured
	3.	M 54	Acute obstruction	Enterotomy	Died
1920	Hayes	F 68	Chronic obstruction	Reduction	Cured
1921	Crescenzi	F 43	Acute obstruction	Reduction	Cured
1923	Thomson	F —	Acute obstruction	Reduction	Cured
1923	Rendle Short	M 20	Many attacks of obstruction	Reduction	Cured
Hernia of Mesentericoparietal (right duodenal) Fossa.					
1924	Novak & Sussman	M 34	Acute obstruction	Reduction	Cured

Continued on next page.

Table I.—Cases of Retroperitoneal Hernia Producing Symptoms Recorded since 1914—*continued.*

Date	Reporter	Sex and Age	Description	Treatment	Result
Retrocolic Hernia.					
1918	Finsterer	M 33	Discovered strangulated, in course of operation for strangulated inguinal hernia	Reduction	Cured
1920	Pribram	M 51	Acute obstruction	Resection of 235 cm. of ileum	Cured
1921	Zoepffel	M 18	Found during operation for appendicitis. As big as child's head	Not reduced	Cured
1923	Copenhaver	M 25	—	—	Died
Hernia into Foramen of Winslow.					
1918	Rawlence	M 18	Acute obstruction; tympanitic tumour	Reduction	Cured
1919	Engstad	M —	Acute obstruction; tense mass in epigastrium	Reduction	Cured
1919	Schmilinsky	F 65	Obstruction for 3 weeks. Cæcum and ileum in foramen	Reduced	Cured
1923	Copenhaver	F 56	—	—	Died
Intersigmoid Hernia.					
1915	Black	F 34	Followed a fall; signs of partial obstruction	Reduction	Cured
1918	Taylor	M 27	Acute obstruction	Reduction	Cured
1923	Carling & Jones	M 47	Acute obstruction	Reduction	Cured
Hernia through the Transverse Mesocolon.					
1919	Pringle				
	1.	F 55	Symptoms like gastric ulcer. Bowel entered lesser sac through transverse mesocolon, and escaped through gastrohepatic omentum	Reduction	Recurred & died
	2.	F 26	Ditto	Reduction	Cured
	3.	M 36	Followed old gastrojejunostomy. Bowel entered by transverse mesocolon, and escaped through gastrocolic ligament	Reduction Reduction	Cured Cured
	4.	F 35	Had also a gastric ulcer	Gastrojejunostomy	Cured
Hernia into Broad Ligament.					
1917	Fagge	F 61	Acute obstruction	Reduction	Relieved
	,,	F 49	Acute obstruction	Reduction	Cured
1920	Barr		Acute obstruction		
1924	Pidcock	F 35	Acute obstruction	Resection of gut	Cured
Anomalous Retroperitoneal Herniæ.					
1914	Primrose	M 60	Found in course of operation. Sac entered beneath middle colic artery	Reduction	Cured
1920	Olmsted	F 25	Symptoms like cholecystitis. Sac entered beneath left colic artery	Reduction	Cured
1923	Carling & Jones	M 31	Recurrent attacks of pain. Sac entered beneath ileocolic artery	Reduction	Cured
1924	Pidcock	M 37	Acute obstruction. Sac entered beneath right side of superior mesenteric artery	Reduction	Cured

Recorded Cases of Retroperitoneal Hernia producing Symptoms during Life.—

Left Paraduodenal Fossa.—Moynihan gives 8 case reports of patients successfully operated on (by Sonnenberg, Tubby, Priestly Leach, Narath, Haberer, Lawford Knaggs, McArthur, and Sherren). At least 10 others died, with or without operation.

The author in 1915 reported 10 further cases. These included those operated on by Vautrin (2), Crisler, Heller, Felten, Von Holst, Obrastzow, Pikin, Gobeit, and Pybus. In Priebatsch's case, the patient died of obstruction, but there was no operation. All the others recovered except Obrastzow's. In Barker's case the condition was discovered accidentally at an operation for cancer of the stomach.

The present report adds 15 fresh cases, whereof 11 recovered after operation, and 4 died.

The total recoveries therefore amount to 27, and the deaths to 16. No doubt many fatal cases go unreported, and there are some further recorded cases scattered about in literature which is inaccessible here.

Right or Mesentericoparietal Fossa (Waldeyer's).—Moynihan gives 1 case successfully operated on (Neumann's), and at least 3 others dying of the condition (Barr's, Paton's, and Zwaardemaker's). The author added 1 case (Haasler's), which was operated on but died of obstruction. Four more old cases (Quénu, Clarke, Rose, Selby) have been added to the list by Nagel; all died. Also 3 more recent cases, described by MacAllum and Miller in 1908 (male, aged 41, died after operation); Mueller in 1911 (male, aged 47, died after operation); and Carson (male, aged 29, recovered after operation). To these Novak and Sussman add an eighth, a man, aged 34, who recovered. Thus we know of 3 recoveries and 10 deaths.

Pericæcal Fossæ.—The commonest of these is the *retrocolic fossa*. In Moynihan's series, there were 6 patients with symptoms, described by Rieux, Aschoff, Mansell Moullin, Atherton, Neumann, and Funkenstein. Of these, 4 died, and 2 recovered (Aschoff, Neumann). To these the present writer added 3, reported by Matthews, Marsh, and Ransohoff. Of these, Matthews' patient died of obstruction without operation; Marsh's and Ransohoff's were cured by operation.

A valuable paper by Pribram contributes 5 more old cases, between 1896 and 1911, all operated on and recovering (Korte, Bonsdorff, Marion, Joas, Krinsky). Since 1914, 3 further cases have been recorded by Pribram, Zoepffel, and Finsterer. All these were successfully operated on. Thus the retrocolic hernias recovering after laparotomy number 12, with 5 reported deaths. Copenhaver mentions another fatal case at the Mayo Clinic, but without details.

Hernia into the *ileo-appendicular fossa* has been described on several occasions, of which Moynihan accepts 5 in which obstructive symptoms were present (cases of Little, Partridge, Nasse, Riese, and MacEwan.) The last two were operated on and recovered; Nasse's case was also operated on, but died. We have no further cases to add.

Hernia into the *fossa of Hartmann* is rare; the only known case is that of Snow, which was fatal. A case found post mortem has been described by

Fleming, but it produced no symptoms during life. The present writer in 1915 described a personal case, in which the entrance to the sac lay to the outer side of the cæcum, looking to the right (*Fig.* 295). This patient recovered after operation.

Foramen of Winslow.—In Moynihan's series, there were 9 cases with symptoms (described by Blandin, Moir, Majoli, Square, Treves, Neve, Adjaroff, Delkeskamp, and Mori). The last 5 were operated on; 2 recovered, in the hands of Neve and Delkeskamp.

In 1906, Jeanbeau and Riche published a monograph on the subject, and reported additional cases from the literature. Their own case is rather doubtfully an example of this condition, and proved fatal though operated on. In addition to this and to those in Moynihan's list, they record 6 cases operated on (by Gangolphe, Rehn, Picado, Stecchi, Reynier, and Marten). Of these, all were fatal except Marten's. Novello published a fatal case, not operated on.

Between 1906 and 1915, I found records in the literature of 4 more, reported by Carwardine, Haw, Morton, and Sinclair. Sinclair's patient recovered. Haw's patient died without operation; the other two were explored.

Since 1914, 3 further cases have been published, all operated on and all recovering (by Rawlence, Engstad, and Schmilinsky). Thus altogether there are 24 cases: 6 were not operated on but died of strangulation; 11 died in spite of operation; and 7 survived. Copenhaver mentions a fatal case at the Mayo Clinic, but gives no details.

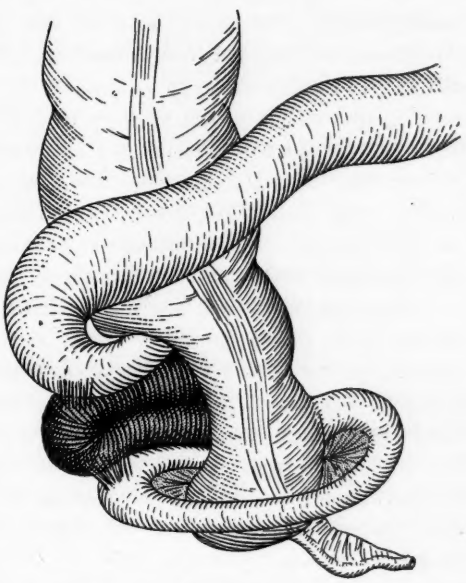


Fig. 295.—Fossa external to the cæcum. (Author's case.)

Intersigmoid Hernia.—Moynihan rejected the oft-quoted case of Jomini, but allowed 2 examples of the condition (under the care of Eve and Eccles). Both died, the first without and the second with operation. To this Coley's case is to be added, which also died without operation.

In addition to these, 2 examples have come to light which were overlooked, both published in 1911, by Krall and Macholl respectively. Krall's case was a man, age 33, suffering from subacute intestinal obstruction, operated on and cured. Macholl's patient, with more acute symptoms, died after laparotomy (*see Fig.* 294).

Since 1914, 3 cases have been published, all cured by operation. One of them (Black's) followed a fall, and showed chronic obstructive symptoms; the diagnosis was suggested before operation. The other two cases (Taylor, Rock Carling and Jones), had acute obstruction.

Thus we know of 8 cases, of which 4 were saved by laparotomy.

Hernia through the Transverse Mesocolon.—In this condition, which is not in quite the same category as those which we have been considering, the hernia does not pass into a congenital peritoneal sac or one formed by stretching, but there is a slit or tear in the transverse mesocolon, on account of its tenuity. It is well known, of course, that rents may occur in the mesentery as a result of grave trauma, but we shall not deal with such. In the described cases of hernia through the transverse mesocolon, there was no history of trauma.

Moynihan does not give any examples of this variety, unless Astley Cooper's stillborn infant can be classed here.

The writer, in his previous communication, quoted 4 examples (those of Mayo (2), Stolzenberg, and Halpenny).

In 1919, Pringle described 4 more, but as one of these had had a previous gastrojejunostomy it does not come into our classification. In his first two cases, the symptoms resembled those of gastric ulcer (in the second there had been hæmatemesis); but at operation a hernia was found of small intestine passing through the transverse mesocolon into the lesser sac behind the stomach, and emerging through the gastrohepatic omentum. The bowel was extracted by operation, with success, but one of the cases recurred and died. In the fourth case, a gastric ulcer was present, but the jejunum was herniated into the lesser sac and emerged through the gastrocolic omentum. Gastrojejunostomy was performed, and a good result obtained.

Hernia into the Broad Ligament.—No record of this condition is known to us prior to 1917, when Fagge described 2 cases, both coming under treatment for acute intestinal obstruction. In the first, there was a loop of ileum passed from behind forward into a hole in the left broad ligament below the ovarian ligament. No fossa is recognized by anatomists in this situation. The patient recovered after laparotomy and reduction, but had further trouble from adhesions. In the second case, the coil of ileum was strangulated in a pouch above the ovary on the right side, entering from behind forwards. This case was cured.

Barr has described another example, of which details are not accessible to me. A fourth case is recorded by Pidcock. The woman had been delivered of a child a fortnight before, and was seized with acute intestinal obstruction. The obstructing band was the round ligament, and the fossa lay below it. Eight feet of gut had to be resected, and there were suppurative troubles, but she eventually recovered.

Anomalous Retroperitoneal Herniæ.—A case of hernia into Fromme's fossa, which lies behind the symphysis pubis and in front of the bladder, was recorded in 1908, and recovered. Possibly there are two other cases recorded by Trñka, but the paper is not available.

Primrose relates finding, during an operation for hour-glass stomach, a large retroperitoneal hernia occupying a thin flabby sac chiefly in the left lumbar region, extending deep into the pelvis, with the opening bounded anteriorly by the middle colic artery, which arose from the right common iliac artery, and proceeded up to the transverse colon. As there were no symptoms, nothing was done. In Olmsted's case the symptoms were diagnosed to be due to cholecystitis, but laparotomy revealed instead a large

flaccid retroperitoneal hernia extending into the pelvis, which contained more than half the small intestines, with the ascending branch of the left colic artery lying in the free falciform edge of the sac. The patient was cured.

Rock Carling and Jones described a case in which there were recurrent attacks of pain, and at operation it was discovered that a loop of ileum had passed into a pouch in the terminal part of the mesentery of the ileum. There was a slit-like aperture 5 inches long, with the ileocolic artery to the right and the last branch from the superior mesenteric to the ileum to the left. A very similar condition was described by Treves in his 1885 Hunterian Lecture. The patient made a good recovery.

In Pidcock's case, the patient was operated on for acute obstruction, and a coil of small intestine was found strangulated in a pouch in the root of the mesentery, entering from the right, having the superior mesenteric artery in front of the opening, and the structures of the posterior abdominal wall behind. It was analogous to a mesentericoparietal hernia of Waldeyer, but entered from the right side of the mesentery instead of the left. Operation was successful.

It is worthy of notice, in most of the varieties of internal hernia, what an important part is played in the production of the sac by vascular bands. These offer a resistance along a particular line, and the peritoneum gets pushed in beneath this line. Thus in the right and left paraduodenal herniæ, the superior mesenteric artery and inferior mesenteric vein respectively act in this way, and the anomalous cases just mentioned show the same feature.

Clinical Features of Retroperitoneal Hernia.—When retroperitoneal hernia produces sufficient symptoms to become of clinical importance, the picture may conform to one of three types: (1) Acute strangulation; (2) Recurrent subacute strangulation; (3) Palpable tumour formation.

If the symptoms and signs are merely those of acute intestinal obstruction, exact diagnosis is of course impossible. In a certain number of the cases, however, as in that which stands at the opening of the present communication, the diagnosis is possible, and, *inter alia*, was made in this particular instance. My patient had had forty or fifty attacks of subacute intestinal obstruction, which were in the habit of clearing up after two or three days. This could scarcely be due to any other cause.

Again, either during an attack of acute obstruction, or apart from any severe symptoms, there may be a palpable tumour in the abdomen, which has several times been mistaken for an ovarian or pancreatic cyst. The characters of this tumour are fairly distinctive, as Moynihan points out. It is limited to a definite area of the abdomen; it is resonant on percussion; distinct gurgling sounds may be heard over it on auscultation; its size and tension vary with the symptoms.

The clinical picture in patients with a hernia through the transverse mesocolon is quite different, resembling those of gastric ulcer.

Treatment.—The operative treatment of these cases may be very easy, or difficult to impossible. As the records show, the results during the past ten years have been much more favourable than in the earlier cases, no doubt

on account of earlier operation. If the gut is already gangrenous, it must of course be resected, and even so several have recovered. Usually the patient is too ill to attempt a closure of the mouth of the sac; in favourable cases this ought to be done, but with due regard for the vessels so often met with in the neck of the sac. If reduction is impossible, it is generally feasible to cut the neck of the sac in an avascular region. When the hernia involves the foramen of Winslow, of course this cannot be done, and the suggestion made by Moynihan is probably the most hopeful; he advises tearing into the lesser sac through the gastrocolic omentum, and incising and emptying a coil or two of the incarcerated gut. After this has been done and the incision in the gut sutured, it will perhaps be possible to withdraw it through the foramen.

TABULAR SUMMARY.

The following cases of retroperitoneal hernia producing symptoms during life are on record, besides a few published in inaccessible communications:—

Nature of the Hernia	Total Cases	Died		Recovered after Operation
		Without Operation	After Operation	
Left paraduodenal fossa	43	—	16	27
Right ditto	5	3	1	1
Retrocolic fossa	17	1	4	12
Ileo-appendicular fossa	5	2	1	2
Fossa of Hartmann	1	1	—	—
Other pericæcal fossæ	1	—	—	1
Foramen of Winslow	24	6	11	7
Intersigmoid fossa	8	2	2	4
Through transverse mesocolon ..	17	—	—	6 +
Into broad ligament	4	—	—	3
Other fossæ	5	—	—	5

I am indebted to Sir Berkeley Moynihan for permission to use *Figs.* 290, 291, and 292.

BIBLIOGRAPHY.

Moynihan and Dobson, *Retroperitoneal Hernia*, 2nd ed.
Rendle Short, *Brit. Jour. Surg.*, 1915, iii, 48, 569.
Andrews, *Surg. Gynecol. and Obst.*, 1923, Dec., 740.
Copenhaver, *Arch. Surg.*, 1923, vii, 332.

Duodenal Fossæ.

Davis, *Boston Med. and Surg. Jour.*, 1914, clxx, 272.
Nazum, *Trans. Chicago Pathol. Soc.*, 1914–15, ix, 191.
Pringle, *Glasgow Med. Jour.*, 1916, lxxxvi, 65.
Schilling, *Kristiania kir. for. f.*, 1916, 68.
Wagner, *Deut. Zeits. f. Chir.*, 1916, 497.
Bramlett and Ashhurst, *Amer. Jour. Med. Sci.*, 1917, clii, 641.
Filauro, *Riv. osped.*, 1917, vii, 59.
Nixon, *Ann. of Surg.*, 1917, lxvi, 233.
Desjardins, *Ibid.*, 1918, lxvii, 195.

GRAD, *Amer. Jour. Obst. and Gynecol.*, 1918, lxxvii, 102.
HALLOCK, *Surg. Gynecol. and Obst.*, 1918, xxvi, 473.
DAYTON, *Johns Hop. Hosp. Rep.*, 1919, 293.
HABERER, *Wien. med. Woch.*, 1919, lxix, 825.
HAYES, *Med. Press and Circ.*, 1919, 279.
RENEKE and LORENZ, *Deut. Zeits. f. Chir.*, 1920, clx, 1.
HARTTUNG, *Ibid.*, clvii, 340.
SCHEELE, *Zentralb. f. Chir.*, 1920, xlviii, 188.
CRESCENZI, *Arch. ital. di Chir.*, 1921, iii, 421.
PHILLIPS, *Bull. Ayer Clin. Lab. Penn. Hosp.*, 1922, 89.
THOMSON, *Brit. Med. Jour.*, 1923, ii, 597.
NAGEL, *Collected Papers Mayo Clinic*, 1923, 135.
NOVAK and SUSSMAN, *Jour. Amer. Med. Assoc.*, 1924, May, 1664.

NOT CONSULTED—
HAEGGSTROM, *Finska lak-sallsk handl.*, 1919, 867.
KUMMER, *Jour. de Radiol. et d'Electrol.*, 1921, 362.

Pericæcal Hernia.
FISHER, *Lancet*, 1914, ii, 145.
FINSTERER, *Wien. klin. Woch.*, 1918, xxxi, 25.
PRIBRAM, *Deut. Zeits. f. Chir.*, 1920, cliii, 75.
ZOEPFFEL, *Ibid.*, 1921, clxv, 267.
FLEMING, *Jour. Anat. and Physiol.*, 1923, lvii, 366.

Hernia into Foramen of Winslow.
JEANBEAU and RICHE, *Rev. de Chir.*, 1906, 435.
WORCESTER, *Anat. Record*, 1916, x, 335.
RAWLENCE, *Jour. R.A.M.C.*, 1918, xxx, 332.
ENGSTAD, *Jour. Amer. Med. Assoc.*, 1919, lxxii, 411.
SCHMILINSKY, *Deut. med. Woch.*, 1919, xlv, 477.

NOT CONSULTED—
STONE, *Washington Med. Ann.*, 1918, xvii, 249.
RADOVAN, *Des Hernies étranglées à travers l'Hiatus de Winslow*, Paris, 1919.
CORRY.

Intersigmoid Hernia.
KRALL, *Deut. Zeits. f. Chir.*, 1911, cx, 303.
MACHOLL, *Beitr. z. klin. Chir.*, 1911, lxxvi, 209.
MURPHY, *Surg. Clin. Chicago*, 1914.
NUZUM, *Trans. Chicago Pathol. Soc.*, 1914–5, ix, 191.
TAYLOR, *Lancet*, 1918, i, 873.
CARLING and JONES, *Brit. Med. Jour.*, 1923, ii, 1082.

Unclassified.
PRIMROSE, *Jour. Amer. Med. Assoc.*, 1914, lxiii, 842.
BOLOGNESI, *Clin. Chir.*, Milan, 1917, xxv, 250.
ZANDER, *Berl. klin. Woch.*, 1917, xxv, 250.
FAGGE, *Brit. Jour. Surg.*, 1917, v, 694.
DEUS, *Deut. Zeits. f. Chir.*, 1919, cli, 222.
PRINGLE, *Glasgow Med. Jour.*, 1919, xci, 129.
OLMSTED, *Amer. Jour. Obst. and Gynecol.*, 1920–1, 273.
CARLING and JONES, *Loc. cit.*
PIDCOCK, *Brit. Med. Jour.*, 1924, i, 369.

NOT CONSULTED—
TRŇKA, *Casop. lek cesk. v. Praze*, 1913, lii, 873.
PILCHER, *Year-Book, Pilcher Hospital*, 1913.
ESPACH, *U.S. Nav. Med. Bull.*, 1916, x, 108.
VAN HOLST, *Upsala Läkeref. Förh.*, 1915–6, xxi, 583.
MATTHEWS, *Northwest Med.*, Seattle, 1916, xv, 372.
BARBER, *Long Island Med Jour.*, 1920, xiv, 268.
BARR, *S. Texas Med. Rec.*, 1920–1, xiv, 5.
FRANK, *Mil. Surgeon*, 1921, xlix, 463.

THE RESULTS OF PERINEAL EXCISION IN THE TREATMENT OF CARCINOMA OF THE RECTUM:

ANALYSIS OF 143 CASES.

By W. B. GABRIEL, London.

The present series of 143 cases represents the total number of perineal excisions of the rectum with colostomy which have been performed in St. Mark's Hospital from 1910 to July, 1924. This paper is intended to give a precise account solely of the results of this operation, immediate and remote, corrected to July, 1924. No case of other modifications of perineal excision has been included in this series, such as perineal excision with preservation of sphincters or formation of a sacral anus.

The type of operation has been that described by Lockhart-Mummery,[1] and may briefly be summarized as follows: A preliminary permanent colostomy is established, and seven to fourteen days later the perineal excision of the rectum is performed. The anus having been closed by a silk purse-string suture, an incision is made round it and is extended posteriorly; the coccyx is disarticulated and the levatores ani are divided with scissors well away from the bowel. The rectum is dissected from the structures anteriorly, using straight scissors and gauze dissection, and the peritoneum is opened anteriorly. The mesorectum is stripped up from the sacrum posteriorly, and is finally divided down to the bowel as high as long forceps and scissors can reach. The bowel is then mobilized sufficiently to enable it to be pulled down and divided well above the growth. The bowel is invaginated with several layers of catgut sutures, and the peritoneum is sutured to the stump, leaving the closure of bowel extraperitoneal. The perineal wound is closed with, or sometimes without, drainage.

The majority of the excisions have been performed under spinal anæsthesia, with scopolamine and morphia, or gas and oxygen.

MORTALITY.

Immediate Mortality.—Under this heading is included every case in which death occurred in hospital following the operation. Out of the total of 143 cases there have been 22 deaths, i.e., 15·4 per cent immediate mortality. During the course of the last three years the operation mortality has been reduced to 12·0 per cent (7 deaths in 58 operations: *see Table IV*). The distribution of deaths between males and females, and the particular cause of death are set out in *Tables I* and *II*.

The increased risk of operation in males as compared with females appears clearly (18 per cent compared with 11 per cent).

Table I.—Operation Mortality.

	No. of Cases	No. of Deaths	Mortality
			Per cent
Males	89	16	18·0
Females	54	6	11·0
Total ..	143	22	15·4

Table II.—Operation Mortality: Cause of Death.

Cause	Male	Female	Total
Sepsis	7	4	11
Urinary fistula	3	1	4
Pneumonia	2	–	2
Shock	1	1	2
Intestinal obstruction ..	1	–	1
Exhaustion	1	–	1
Cause indefinite	1	–	1
Total ..	16	6	22

Local sepsis, with death from toxæmia or septicæmia, occurred in 11 cases. There were 4 fatal cases of urinary fistula: 3 were in men, in whom the fistula was in connection with the bladder or urethra, and 1 was in a woman whose left ureter was injured, with subsequent ascending pyelonephritis. Post-operative pneumonia accounted for 2 fatal cases. There were 2 deaths from shock in this series. One case was that of a female, age 45; severe hæmorrhage occurred in the course of the operation, and she died a few hours later. The other was a man, age 58, whose growth was found at operation to be adherent to the sacrum; a large portion of bone had to be removed with the growth, and persistent hæmorrhage occurred in the course of a difficult operation; in spite of blood transfusion he went down-hill and died twenty-four hours later. One case of intestinal obstruction in a male subject occurred three days after perineal excision. His abdomen was explored, and a loop of small gut was found to be strangulated in the internal foramen formed between the abdominal wall of the left iliac region and the colon as it came forwards to the left hypogastric colostomy. The loop of small intestine was viable and was freed, but the patient did not survive. One man died from exhaustion for no definite cause a month after operation, and in one case in which death occurred on the third day the notes are defective.

Ultimate Mortality.—Of the 121 patients discharged from hospital convalescent after perineal excision 44 are known to be dead. An analysis of their duration of life after operation shows that when recurrence takes place it causes death, as a rule, inside 5 years after operation (93 per cent), and that 80 per cent of these deaths occurred inside 3 years. *Table III* makes this clear:—

Table III.—Table of 44 Subsequent Deaths, showing Duration of Life after Operation.

Under 1 Yr.	1–2 Yrs.	2–3 Yrs.	3–4 Yrs.	4–5 Yrs.	5–6 Yrs.	7–8 Yrs.	Indef.
12	17	6	3	3	1	1	1

A consideration of the cause of death, so far as can be ascertained, shows that recurrence in the pelvis occurred in 11 cases, in the abdomen or liver in 11 cases, and 2 are alleged to have had secondary deposits in the brain. In 16 cases the cause of death has not been ascertained, while in 4 cases the cause was probably unrelated to the carcinoma of the rectum: thus cerebral hæmorrhage is stated to have brought about the death of 2 patients who were otherwise well; one female developed a highly malignant and rapidly fatal carcinoma of the breast between 4 and 5 years after operation. The case of the man who died in the 7th year after perineal excision has been described by Lockhart-Mummery[2]: in the 4th year after excision a constricting carcinoma developed in the blind colonic segment well away from the pelvic floor, without any evidence of secondaries in the pelvis or abdominal glands; it was considered to be probably a new formation, not related to the rectal growth; this second carcinoma was excised, but the patient died 3 years later with extensive deposits in the liver, aortic glands, and glands in the neck.

A consideration of the 35 deaths occurring in less than 3 years after operation shows that in 15, i.e., 43 per cent, there was extra-rectal spread at the time of operation. Thus in 5 cases a mass of glands and thickened lymphatics was found in the mesorectum, and death occurred from recurrence in the liver in each case at intervals varying from 10 months to 2 years 4 months; in 5 cases the growth had involved prostate, bladder, vagina, or the tissues posteriorly; 2 cases were associated with abscess formation, one a peri-anal abscess, the other a high-lying post-rectal abscess which was discovered at operation, evidently due to perforation of the rectal wall at the base of the ulcer; in 2 cases the growth had extended down to the anus; in one case, what was thought to be a small, secondary nodule was felt in the liver when the preliminary colostomy was done, but it was decided to perform the excision in spite of this. Many of the remaining 20 cases, though movable, presented extensive and deeply ulcerated tumours, and in 2 of the cases the growth was several inches above the peritoneal reflection, rather beyond the scope of a perineal excision.

In every case in which recurrence occurred in less than one year after operation, some local cause such as those just mentioned had been found to account for so rapid a recurrence.

Cases Untraced.—Thirteen cases are untraced, of whom 11 were operated upon more than 5 years ago. It is possible that some of these may be still living, because in 1920-22 three were traced and known to be living free from recurrence for periods of 9, 4, and 3 years respectively: they

have subsequently been lost sight of. One case was known to be recurrent in 1919 when last heard of.

ANALYSIS OF CASES.

Cases Now Living After Perineal Excision.—By deducting from the total 143 cases 22 immediate deaths, 44 subsequent deaths, and 13 untraced, the figure is arrived at of 64 patients who are now living after perineal excision.

Table IV shows the cases grouped into three main divisions: (1) Those operated upon within the last 3 years; (2) Those operated upon between 3 and 5 years ago; (3) Those operated upon more than 5 years ago. The table is an important one, and from it can be easily calculated the operation mortality in the different periods, and the percentage of cure.

Table IV.—Cases Grouped according to Length of Time which has now (July, 1924) elapsed since Operation: Indicating Percentage of Cure.

	Less than 3 Years after Operation	Between 3–5 Years after Operation	More than 5 Years since Operation	Total
No. of cases	58	22	63	143
Immediate mortality ..	7	6	9	22
Subsequent mortality ..	8	9	27	44
Untraced	1	1	11	13
Alive with recurrence ..	3	1	1	5
Alive and well	39	5	15	59

So far as is known as the result of a very recent inquiry, these 64 cases are all well and free from recurrence with the exception of 5; 3 of these are under 3 years' duration, recurrent in perineum, pelvis, and liver respectively: one is a man who now, 4 years after operation, has irregular hæmorrhage and discharge from the distal colostomy opening, which is most probably due to recurrence in the intrapelvic portion of the colon, and the last is a man 6½ years after operation in whom the growth has definitely recurred in the segment of colon distal to the colostomy, together with secondary deposits in the liver.

To refer now to the cases alive and well at the present time, it will be seen from *Table IV* that there are 39 cases less than 3 years after operation, 5 between 3 and 5 years, and 15 more than 5 years since excision. These last are the most important.

There are two cases free from recurrence 13 years after perineal excision: one is a lady now 82 years old, who is still able to do some house-work; the other is a man of 72 who, until two years ago, did his work as a sewer foreman. There is one case alive at 10 years, one at 9 years, two over 8 years, and 9 cases alive and well at periods of 5 to 8 years after operation.

The functional result in most of these cases is extremely good. Thus, to consider the 20 cases of over 3 years' duration free from recurrence, we

find the following results: 6 are women, of whom 5 are able to do their housework, and one works as a stationer's assistant. Of the 14 men under consideration, 10 perform such varied work as the following occupations suggest: printer's cutter, fish-curer, schoolmaster, packer, tailor, pilot, messenger, waiter, plate-layer (age 63), and one does odd jobs. Three are living in the ease of retirement, being now 65, 71, and 72 years old respectively, and one is a stout man of 59 who used to be a farm labourer, but no longer works.

Age.—Advanced age has not been considered in itself to contra-indicate perineal excision. Of these 143 cases, no fewer than 37 were between 60 and 70 years of age, and 4 were over 70. The oldest patient operated upon was a woman of 76 upon whom Mr. Graeme Anderson performed a perineal excision in September, 1923; she is at present in good health and carries on light household duties.

The immediate mortality in the 41 cases over 60 years of age was 8 (20 per cent), and 19 of them are at present alive and well for periods of up to 8 years.

The cases under 30 years of age who have been operated upon have, as usual, done badly. There were 3 of these, of ages 26, 28, and 29. Two died in less than 3 years after operation, and the third has never been traced.

Microscopical Examination.—In the earlier cases in this series histological examination was, unfortunately, only seldom carried out. While all the cases done in recent years have been examined, only 8 of the 22 cases living more than 3 years have a microscopical diagnosis of carcinoma. The clinical diagnosis, however, remains, and is not lightly to be queried: at a special hospital like St. Mark's a great wealth of cases is seen, and experience shows that a diagnosis of cancer by sight and touch is only very rarely mistaken. Furthermore, the records of many cases not sectioned are rendered the more valuable in that good macroscopic descriptions of the specimens are available and support the clinical diagnosis of malignancy.

This point is now receiving particular attention, and care is being taken to have a complete examination made of every specimen removed.

Operability Rate.—In the period under review, 1910–24, 521 new cases of cancer of the rectum and colon were admitted into hospital, of which 230 were operable; this gives an operability rate of 44 per cent. If corresponding figures are taken for the last five years, the operability rate is found to rise to 54 per cent (116 operable out of 216). This latter figure may be somewhat in excess of the true figure, because a certain amount of judicious selection has to be exercised in admitting cases; there is a limit to the number of colostomy cases that can be dealt with by the nursing staff at one time, and it is not sound policy to fill up available beds with hopelessly inoperable cases while there are operable and borderline cases urgently requiring admission. It is our practice to admit for further examination and exploration every case in which there appears to be the remotest possibility of the growth being excisable. Probably about 50 per cent is a good estimate of our present operability rate.

The following three cases are examples of the advanced conditions of growth which have been considered as operable. They show what brilliant

results may be obtained by perineal excision for cancer in the middle and lower thirds of the rectum.

Case 1.—F. R., male, age 57, 1918, under the care of Mr. Lockhart-Mummery. He presented an ulcerated growth low down on the anterior rectal wall. Preliminary colostomy was done on Dec. 2, 1918, followed a week later by perineal excision. The prostate was found to be involved, and a portion of the prostate, together with the seminal vesicles, was removed with the growth; the bladder was opened and sutured with catgut. The patient made a good convalescence without any urinary leak; since then he has remained in good health, and in a letter dated June, 1924, he states that his health is good and he does light work as a packer; his colostomy is satisfactory, the wound is healed, and there is no sign of recurrence.

Case 2.—H. J., male, age 49, 1919, under Sir Charles Gordon-Watson. He was a stout, florid-looking subject, with a hard irregular growth in the posterior wall of the rectum extending down to the anus; his inguinal glands on the right side were enlarged and hard. A preliminary colostomy with a gland dissection of the right groin was performed on April 30, 1919, and on May 5, a perineal excision was done. He made a satisfactory recovery, and was discharged on June 7th. The growth was proved to be a columnar-celled carcinoma with secondaries in the inguinal glands. Since then he has carried on his former work as a bank messenger, and in a recent letter he states that his health is good, and he is doing his work, that his wounds are healed, and that there is no sign of return of the growth.

Case 3.—W. B., male, age 66, in March, 1920, under the care of Mr. Lockhart-Mummery. He seemed old for his years, and in a poor state of health generally; he was a waiter. He presented an extensive malignant growth in the middle third of the rectum, and had been sent in from out-patients as a border-line case for examination. It was decided that the growth was removable, and he was treated by colostomy and perineal excision. He subsequently developed a urinary fistula, probably from sloughing of the base of the bladder, but after a stormy convalescence this healed and he recovered. Since then he has resumed his work as a waiter, being now 70 years old. He looks extremely well, and a recent examination showed his perineal wound is soundly healed and that there is no sign of recurrence.

Percentage of Cure.—In estimating the percentage of cure, all cases done less than 3 years ago must be excluded. In *Table V* are shown the percentage of 3- and 5-year cures, the figures being arranged in two divisions calculated on (*a*) the total numbers submitted to operation, (*b*) the survivals from operation. This duplication of figures seems to be necessary for clearness owing to the different standards adopted in recording results such as these.

Table V.—Percentage of Cure.

Description	3-year Cure	5-year Cure
a. Figures based on total numbers submitted to operation ..	23·5 per cent (20 out of 85)	24·0 per cent (15 out of 63)
b. Figures based on survivals from operation	28·5 per cent (20 out of 70)	28·0 per cent (15 out of 54)

The remarkable similarity between the figures for 3- and 5-year cures in the two columns will be noted, and might have been expected from a

consideration of *Table III*, which showed that 80 per cent of subsequent deaths after operation occurred inside 3 years. The anomaly in division (*a*) of the figure for 3-year cures being less than that for 5 years is due to the abnormally high operation mortality in the 3- to 5- year period, which point is shown corrected in division (*b*).

In conclusion, I wish to express my thanks to the Medical Research Council for their financial assistance in starting and carrying on the cancer follow-up scheme of which this paper is the firstfruits, and to my colleagues at St. Mark's Hospital for their ever-kindly help and encouragement.

Finally, in publishing these results, a tribute should be paid to the nursing staff of St. Mark's Hospital, many of whom have given long years with devotion and enthusiasm to the nursing of these cases.

REFERENCES.

[1] LOCKHART-MUMMERY, J. P., *Lancet*, 1920, i, 20.
[2] LOCKHART-MUMMERY, J. P., *Proc. Roy. Soc. Med.*, 1922, xv, 38.

CASE OF OSSEOUS METASTASIS FROM PRIMARY CARCINOMA OF THE RIGHT MAMMA.

By Sir GEORGE THOMAS BEATSON, K.C.B., Glasgow.

Although the cause of cancer is still an unsolved mystery, we know that the disease, notwithstanding its local origin, eventually, as a rule, spreads throughout the body, and that it may do so either in a slow and stealthy manner, or it may advance with rapid and giant strides under conditions which seem to favour its progress. To these new manifestations of the disease the term 'metastasis' has been applied, and they are the important element in cancer, being often the main factor in its ultimate fatal result. That metastases vary with the situation of the primary growth is a well-known fact, so that the prognosis in a case of carcinoma is to a great degree affected by the initial seat of origin of the disease. This is well illustrated by the study of the course of primary cancer of the uterus and breast. In 1914, Lazarus-Barlow[1] stated that the experience of the Middlesex Hospital (and it is borne out by my own at the Glasgow Royal Cancer Hospital) shows that 55 per cent of autopsies on cases of carcinoma of the cervix uteri reveal the complete absence of metastasis, while only 3·2 per cent of autopsies in mammary cancer show a similar freedom—a fact that must be a considerable bar to successful radiation against recurrence in these latter cases.

Ignorant though we may be of the exciting cause of cancer, it is no longer a mystery as to how it spreads throughout the body. Clinical and pathological study have elucidated the method by which the infection is carried, and the roads by which it travels. No longer are metastases regarded as evidence of a 'cancerous diathesis', or the outcome of 'cancer juice' diffused through the tissues and organs. They are indications that cancer cells have travelled to the points affected and are behaving as 'secondary tumours'. Whether these detached cellular elements find their way along by 'spontaneous movements' (Roger Williams), by 'permeation' (Handley), or by 'forcible dissemination' (Beatson), is immaterial. What we do know is that the two channels of dissemination are the lymphatics and the blood, and that in some cases both routes are followed at the same time. By Handley, lymphatic dissemination is upheld almost to the exclusion of that by the blood.

Reference has already been made to the frequency of metastases in mammary carcinoma, and an analysis of the seat of their occurrence has been studied by Gross. His table is based on 423 necropsies that he had collected. From it we learn that the pleuræ (50·9 per cent), lungs (49·9 per cent), liver (48·6 per cent), and bones (20·5 per cent) were the organs and structures chiefly attacked. In the case of the bones there exists a considerable variety of opinion as to whether general osseous invasion is common or the reverse, and as to the frequency with which individual bones are attacked. This uncertainty

is probably due to the fact that autopsies on cancer patients are not always complete in connection with the osseous system, so that nothing would be gained by discussing the point. Looking, however, to the view expressed very emphatically by Handley that in mammary cancer secondary bone deposits only occur in bones which lie partially or wholly in the area liable to subcutaneous nodules—in other words, that they are due to lymphatic dissemination and not to blood infection—the following case is of interest.

CASE REPORT.

Mrs. F., age 55, was admitted to the Glasgow Royal Cancer Hospital under my care in August, 1913, complaining of a swelling in the right breast of six years' duration. The family history showed that her mother died at the age of 84, of senile decay, and that her father's death was due to cancer of the tongue. She was one of a family of eight, and had only one brother alive. Two of the brothers died abroad, another from consumption, and one sister from 'a complication of troubles'.

History.—The personal health of the patient had always been good. She had been married for twenty-eight years, but had no family. She never had any trouble with the right breast, and no injury to it. Six years previously she had noticed a lump in it. She consulted a medical man, but nothing was done. The lump gradually increased, giving her, however, no inconvenience until March, 1913—about four months before admission to this hospital—when she felt her right arm less useful and began to suffer from sciatica in her left leg, which caused some difficulty in walking. Apart from these symptoms her general health was good and she had not lost flesh.

On Admission.—The upper quadrants of the right breast were completely infiltrated with the new growth, and it had to a slight extent invaded the lower quadrants. To the feel the growth was very hard and very irregular in shape. It was adherent to the skin, which was bossed in places and discoloured, but was not broken. The nipple was retracted. There were some subcutaneous nodules to the right of the upper part of the sternum, and the right axillary glands were enlarged. There was no swelling of the arm, and no tenderness was complained of in handling the growth. As far as physical examination showed, there were no metastases in either the thorax or abdomen. Careful examination of the spine revealed no tenderness, nor could any be elicited along the left sciatic nerve. The only thing noticeable was a complaint of stiffness in the outer muscles of the left thigh. The urine was acid, and contained neither albumin nor sugar.

The diagnosis was that of carcinoma of the mamma. As the case was not suitable for any attempt to remove the growth locally, and as the possibility of general dissemination of the disease was recognized, no operative measures were undertaken, and palliative treatment only was followed.

During the first two months of patient's residence in hospital nothing special occurred. The chief symptom was a very constant complaint of rheumatic pains in the back and limbs. The case chart shows pulse, respirations, and temperature to have been normal. On Oct. 3 a significant incident happened in the form of a fracture of the right clavicle when the patient was

raising herself in bed. It was treated by rest, and union resulted, as was confirmed at the post-mortem examination. On Oct. 12 bosses were noticed on the scalp, and a skiagram showed implication of cranial bones. The further chief notable incidents in the case were the fracture of the left humerus on March 2, 1914, and of the left femur on March 18. There was also loss of sight in the same month. Skiagrams showed the presence of carcinosis in all the bones of both limbs, and the urine on March 7 revealed Bence-Jones' albumose —a recognized indication of extensive bone involvement. The pathological report of the urine on March 7, furnished by Dr. Whittingham, was as follows: "Acid; sp. gr. 1018; no sugar; no albumin, but a trace of Bence-Jones' albumose." A further report on March 9 gave the amount of urine in twenty-four hours as 1000 c.c., against the normal 1500 c.c, and of 0·018 grm. of urea per c.c. as against a normal 0·02, giving 18 grm. of urea in twenty-four hours against the normal 30 grm., almost half less than the normal amount. The pathological report of the blood on March 4 was as follows:—

Blood-count—

Red blood-corpuscles	3,500,000	Colour index	1
Hæmoglobin	70·0 per cent	White blood-corpuscles	70,000

Differential Count—

Granular series	85 per cent (normal 75 per cent)	Myelocytes	4·0 per cent
		Hyaline series	15 per cent (normal 25 per cent)
Neutrophils	77·0 per cent		
Eosinophils	1·0 per cent	Small mononuclears	11·0 per cent
Transitionals	3·0 per cent	Large mononuclears	4·0 per cent

There were thus present a secondary anæmia and marked leucocytosis, mainly of polymorphs. On April 23, 1914, the patient died, and on the following day a post-mortem examination was made by Dr. Harold Whittingham, to whom much credit is due for the interest he took in this case, and for his valuable reports on it. Without these the case would have been very incomplete and much of its usefulness lessened.

POST-MORTEM REPORT.

The body is that of an emaciated woman. A *tumour mass* occupies the right mamma, extending from the anterior axillary line to within an inch of mid-sternal line transversely, and from 2nd to 6th ribs in the mid-clavicular line vertically. The nipple is retracted. There is slight ulceration in one spot (*Fig.* 296). Surface of growth bossy, and skin over it bluish-red in colour. Growth fixed to chest wall; skin adherent to growth. The skin round the growth for a radius of 3 in. from the nipple contains numerous small shot-like subcutaneous nodules; a few also felt as far round as the posterior axillary line; one between scapulæ.

The right axillary lymph glands are enlarged and hardened. A dense hard tumour mass surrounds the middle third of right clavicle (*Fig.* 297). Right clavicular glands enlarged. Inguinal glands free. Hard bosses felt over bones in frontal, parietal, and occipital regions. The left mamma is free. No œdema of arms or face.

The right axillary and supraclavicular glands are much enlarged and

hardened with dense white growth. In the left axilla the pectoral group of glands is involved.

The subcutaneous fat is scanty and normally pigmented.

The right mamma on dissection shows the breast proper to be replaced by dense white carcinomatous growth, which growth has involved the right pectoralis major muscle, and spread as a dense sheath of growth between the pectoralis major and minor to the costocoracoid membrane, especially in the direction of the coracoid process of the scapula. The growth has also penetrated to the thoracic wall. Thus, on the right side the 3rd, 4th, 5th, 6th ribs,

FIG. 296.—Showing the appearance of the breast post mortem.

and 3rd to 6th intercostal muscles are involved, while on the left side the growth has spread across the fascia covering the sternum, and involved the 3rd to 8th ribs and intercostal muscles. The left breast has, however, escaped.

Thorax. — The *pericardium* is chronically inflamed—'milk-spots' over the base of the left ventricle and the right auricle anteriorly. No carcinoma found here. The *heart* is of normal size; the cardiac muscle is a little pale (diffuse fatty degeneration). Ante-mortem clot in both ventricles, also in the right auricle and pulmonary artery. Valves normal. Slight atheromatous changes in the aortic arch.

Both pleural sacs are more or less adherent all round, especially the right sac. The left sac contains about a half-pint of clear yellow fluid. Small white carcinomatous nodules, varying in size from pin-head to small pea, dotted over both visceral and parietal layers on either side, especially numerous on the right side. Diaphragmatic pleura is thickly studded with these nodules. The spread to pleura is probably lymphatic.

Both lungs are studded throughout with small nodules of growth—whitish, and size of a small pea, and probably spread by the blood. No evidence of spread along the bronchi.

The *anterior mediastinal lymph glands* are slightly involved. Posterior set and bronchial glands are apparently free. *Thoracic duct* appears to be free.

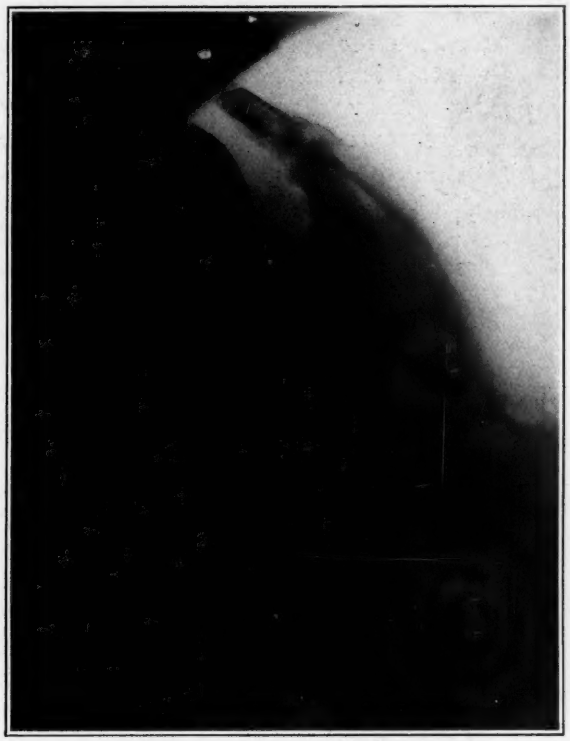


FIG. 297.—Fracture of right clavicle.

Abdomen. — The *peritoneum* contains two small white carcinomatous nodules below the level of the umbilicus and along the course of the right deep epigastric artery. The *liver* is a little enlarged and fatty, with marked perihepatitis. Numerous carcinomatous nodules are scattered throughout the liver both in its substance and near the surface. These nodules vary in size from a split-pea to that of a five-shilling piece, the latter being a nodule in the centre of left lobe of the liver. The nodules in centre are larger than those near the surface. Some are hæmorrhagic and, in places, necrosing. Many of the surface nodules are umbilicated. Spread here may be lymphatic or blood, or both. The larger size of the central nodules compared with those at the surface of liver points to some, at least, arriving through the blood-stream; some show definite relation to portal tracts. The *stomach and intestines* are normal. The *spleen* free from carcinoma.

The *ovaries* are shrivelled and puckered. The *uterus* contains in its interior a large intramural fibroid, the size of half-a-crown, breaking down in its centre. The *bladder* is normal. The *kidneys* are of normal size. Numerous small nodules of growth are dotted throughout them, especially in the cortex, some of the nodules having a definite relation to the blood-vessels. Some are just under the capsule, but even here a definite layer of renal tissue is seen between them and the capsule—apparently blood-spread to kidneys. There is some involvement of the *aortic lymph glands.*

Iliac lymph glands are free of carcinoma, as also are *pouch of Douglas* and *psoas* and *iliacus muscles*. A few slight old adhesions are present in the pouch of Douglas. Calcareous plaques in the *aorta* (*abdominal*) and *common iliacs*.

Osseous System.—The *right clavicle* (*Fig.* 297) is the seat of a healed fracture at the junction of its outer and middle thirds. The union is good, and is surrounded with a callus which is found to consist for a great part of tumour tissue. The centre portion of this right clavicle is also cancerous. The spread of carcinoma here has probably been by costo-coracoid membrane.

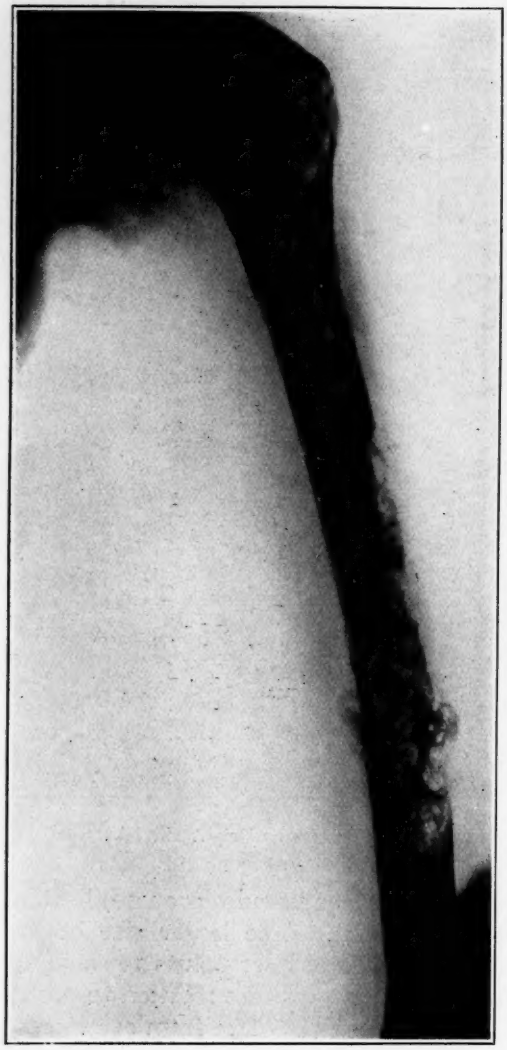


Fig. 298.—Fracture of left humerus.

The *left humerus* (*Fig.* 298) has fractured below the level of the deltoid insertion. It is incompletely united, much tumour tissue uniting the broken ends and surrounding them. On section of the humerus, nearly the whole medullary cavity is found filled with tumour tissue of a yellowish tinge; towards the centre of the shaft some red marrow is seen amongst the tumour tissue. The infection of the bone appears to have been through the marrow, and then to have spread outwards, eating its way to the surface of the bone by pressure atrophy. The spread to bone was probably by the nutrient artery. The various muscles, tendons, and fasciæ going to the humerus were carefully examined, but no evidence of direct spread by this route could be found. The only place where any growth was found in muscles and fascia was around the seat of fracture, and this can be readily explained.

The *right humerus* is also extensively involved in its marrow cavity, but as on the left side no evidence of spread by fascia or tendons, etc., was found.

The *left femur* (*Fig.* 299) is fractured high up in the upper third of the shaft. The union is poor. Much tumour tissue is present in the callus. On section the whole marrow is more or less replaced by yellowish-white tumour tissue, and some red marrow was still seen here and there towards the centre of the shaft. Compact bone much thinned all round. No increase of growth

noticed at the point of muscle insertions, and no evidence of spread along the tendons, fascia, etc. Apparently blood-spread. Some slight infection of vasti muscles at seat of fracture. The *right femur* (*Fig.* 300) is in a similar condition, but no fracture present. The *first left metatarsal bone* (*Fig.* 301) on section shows white nodules of tumour growth in its centre and especially at its distal ends. The *phalanges*—third and fourth middle ones—are in a similar condition. with no sign of tendon spread.

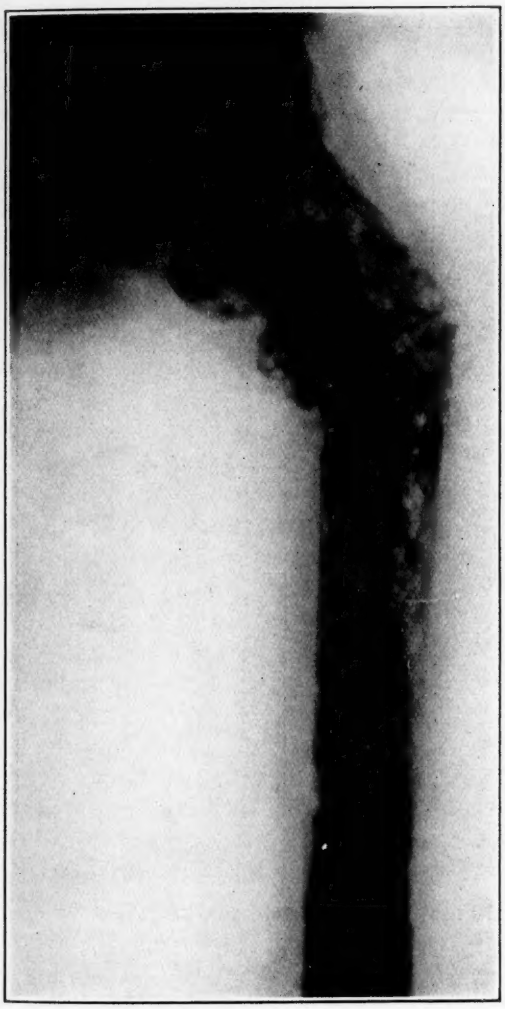


FIG. 299.—Fracture of left femur.

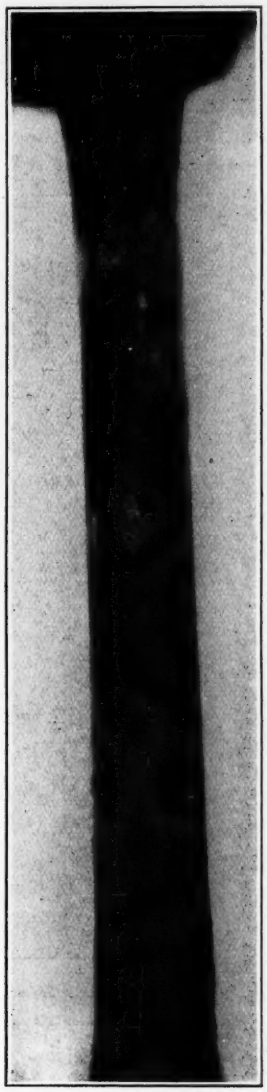


FIG. 300.—Right femur.

The *sternum* is free from carcinomatous involvement, which is strange, as it is often involved in cases of direct or lymphatic spread.

The *3rd to 6th right, and 3rd to 8th left ribs* are involved by direct spread and apparently through the lymphatics.

The *skull* shows fascial spread by cervical fascia, and numerous nodules

lying over the cranial bones and eroding into them. On removing calvarium, varying-sized nodules (5 to 20 mm. in diameter) are also seen between dura and the bone, leading to some compression of the brain, the convolutions being somewhat flattened. At the left sphenoidal fissure a moderate-sized growth involves structures here, and has invaded the sphenomaxillary fossa and back of the left orbit, this, with the cerebral compression, accounting for the optic atrophy. The diploë of the bone of the cranial vault is also involved, here probably from nodules in dura and pericranium, but possibly in addition from the blood. No nodules found in the *brain* or *pia mater*. The optic atrophy on both sides was clearly from cerebral compression.

FIG. 301.—Carcinosis of first metatarsal bone of left foot.

FIG. 302.—Carcinosis of right tibia and fibula.

The *9th to 12th dorsal and 2nd to 4th lumbar vertebræ* were found to be extensively involved in their bodies. The cervical vertebræ were not examined. These infections may have been due to lymph-spread by intercostals —especially as some nodules were seen in the anterior spinal ligament in cervical, lumbar, and dorsal regions. No involvement of the cord was found.

The *radii, ulnæ, tibiæ, and fibulæ* (*Figs.* 302, 303) were all involved, with large nodules and masses of tumour tissue, chiefly in the marrow cavity. In none of these instances could any evidence be found of lymph-spread, and there

was no more tumour tissue at sites of tendon insertions than elsewhere in the bones—in fact, if anything, less. So also in humeri and femurs, and notably in the phalanges examined (*Figs.* 304, 305), where the nodules were most numerous at the distal ends of bones. These points favour blood-spread, though in the case of the ribs, vertebræ, and skull there is evidence of spread by fascial planes and lymphatics.

Sections were made of the mammary tumour, of the infected marrow, of the various bones, and of the scalp, liver, spleen, kidney, and aortic glands. In all of them, save the spleen, there was confirmation of the macroscopic appearances showing that they had originated in a typical spheroidal-celled scirrhus of the mamma.

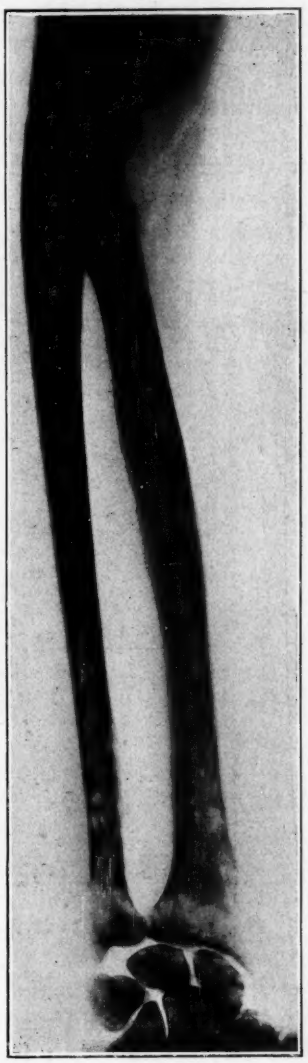


FIG. 303.—Left radius and ulna.

* * * * *

In the above clinical and pathological record there are several points of interest to be noticed. The first one is the loss of sight in March, 1914. This was not due to cerebral metastases, as there was no carcinoma spread to the brain itself. The blindness was evidently the result of optic atrophy on both sides caused by brain compression induced by the varying-sized nodules found lying between the dura and the bone, and by their subsequent passage down to the middle fossa of the skull, causing at the left sphenoidal fissure a moderate-sized growth which involved the structures there and invaded the sphenomaxillary fossa and the back of the left orbit.

Another point is that the case is illustrative of what has been observed in similar ones—namely, a not very active carcinoma becoming the focus of a silent, stealthy, and widespread osseous dissemination of the disease, accompanied by few local manifestations of its presence. Beyond the complaint of indefinite rheumatic pains, especially in the back, there was nothing to indicate what had taken place. Neither local tenderness in the bones nor tenderness over them was present; nor was pyrexia revealed in the form of rise of pulse or temperature. It was not until the occurrence of the different fractures that attention was directed to the osseous system, and it was only when use was made of the X rays that the extent of the bony lesions was revealed, thus clearly indicating the need for a more systematic use of skiagraphy in all cases of carcinoma, especially of the mamma, and, above all, in those that it is proposed to treat by the deep (Erlangen) X-ray therapy.

Another point of interest to be noted in this case is the reparative power apparently possessed by bones which are the seat of carcinosis. This is well illustrated in the case of the right clavicle (*Fig.* 297), which was the first bone fractured. The post-mortem examination showed that it was the seat of a healed and united fracture at the junction of its outer and middle thirds, although the centre of the bone was cancerous. In this instance the spread of the carcinoma was probably lymphatic by the costocoracoid membrane, for at the post-mortem examination it was found that the mammary tumour had spread through the pectoralis major and was seen as dense white growth infiltrated between that muscle and the pectoralis minor and spreading by it

FIG. 304.– Carcinosis of right finger bones.

FIG. 305.—Carcinosis of left finger bones.

up to the costocoracoid membrane and to the outer half of the clavicle, where there was callus (largely mixed with new growth) surrounding the firmly united fracture. In the case of the fracture of the left humerus (*Fig.* 298) and left femur (*Fig.* 299) there was good evidence of an attempt at union, but it was incomplete and poor in comparison with the clavicle. In both cases, however, callus was present, though largely composed of tumour tissue.

It is not easy to dogmatize on the course of events that leads up to fracture in bone carcinosis. There may be a preliminary diminution in the salts of the affected bone ; but it would almost seem as if the presence of the tumour cells in the interior of the bones did not arrest the powers of the bone-forming osteoblasts on their outer aspect, for the callus present in the united

clavicle, as also in the case of the humerus and femur, was a blend of osteogenetic and tumour cells.

In view of the close connection that exists between the red marrow of the long bones and the cellular elements of the blood, it is a very natural question to ask: "What was the blood picture in the case? Did it mirror the condition of the bones? If so, what variations did the formed elements of the blood undergo?" In answer to these queries it may be said that there were very distinct anatomical changes in the blood, and that they are of considerable interest, but they cannot be said to reflect the osseous lesions found by the X rays and confirmed by the post-mortem examination. If reference is made to the pathological report on the blood on March, 1914, seven weeks before death, it shows that there was a distinct diminution of red cells and a great increase of leucocytes, mostly of the polymorph type, with a small percentage of myelocytes; but, beyond this lesser number of erythrocytes, there was no evidence of pathological changes in the shape of nucleated red corpuscles and megalocytes—manifestations one would have expected in such extensive bone-marrow changes. This is a very striking feature in the case. In no way is it in keeping with the blood changes found in what has been called by Piney "the metastatic anæmia of osseous carcinosis".

Harrington and Teacher point out that the recorded cases of osseous carcinosis, secondary to primary carcinoma of the stomach, breast, uterus, bile-ducts, and prostate, fall into two groups—one characterized by no special changes in the blood, and the other by very marked variations of a nature suggestive of pernicious anæmia, namely, high colour index, reduction in hæmoglobin and red cells, and the presence of megaloblasts. It is to the first of these groups that this case must be assigned. As to the conditions that regulate or control the anæmia of cancer, probably the most important are ulceration of the primary growth, sepsis, and hæmorrhage. None of these was an active factor in this case, so there was not present in connection with the bone-marrow any 'erythroblastic reaction' which, with its multiplication of red cells normally present in marrow, turns it from yellow into red. In the present case, sections of the right humerus and femur showed the epiphyseal ends of the bones pale and somewhat yellowish, due to the presence of a large amount of tumour tissue, proved to be such by the microscope. The diaphyses contained a little red marrow, but their striking feature was a large amount of whitish and moderately dense tumour tissue. It will thus be observed that the red marrow is absent from the epiphyseal ends of these bones—its normal situation—and that the little red marrow present was seen in the shaft of the bone. The microscopic examination of the tumour tissue in the marrow of the bones showed it to consist of typical scirrhus carcinoma, identical with the condition found in the mammary growth. This latter consisted of strands and clumps of spheroidal epithelial cells lying irregularly amongst a dense and abundant fibrous stroma. In short, the growth in the bones was a typical scirrhus carcinoma, and it had for the greater part replaced the ordinary bone-marrow, but any remains of the yellow marrow showed little change from normal. The first metatarsal bone on the left side (*Fig.* 301) furnishes an illustration of the whole of the contents of the medullary cavity being replaced by tumour tissue.

Interesting as the above-mentioned points are, they are of minor value compared with the importance of this case as a contribution to the still contentious point as to the channel by which osseous carcinosis takes place. Since the abandonment of the 'cancer-juice' explanation of the spread of cancer through the body, two theories as to cancer dissemination have prevailed, namely, the 'embolic' by the blood, and the 'lymphatic' by the lymphatics. In the former, particles of the primary carcinoma find their way into the blood and are distributed by its current to different parts of the body, where the transmitted cells proliferate and produce secondary nodules, the channels of invasion being either the thoracic duct or the small veins in the vicinity of the primary tumour. In either case, the particles must pass through the lungs to reach the systemic circulation. In the lymphatic mode of spread, the extension takes place by the passage of the cancer cells along the lymphatics of the part, thus enabling them to invade neighbouring areas. As to the respective frequency of these two modes of dissemination, the former view was that the embolic process was responsible for the largest number of cases, and in F. von Recklinghausen it found a strong exponent. In late years another supporter of it has been M. B. Schmidt, of Jena. But to several workers its acceptance presented difficulties, one of the first men to take exception to it being Mr. Stephen Paget[2], who, as far back as 1889, dealt with the subject in a paper published in the *Lancet*. It would serve no good purpose to give in detail the arguments put forward by Mr. Paget, as the work in recent years of Mr. W. Sampson Handley, of the Middlesex Hospital, has made it clear that the usual path of the cancer spread leading to metastases is by the lymphatic system, that embolic dissemination is less common, and that it is not necessary to have recourse to it to explain the osseous and other distant metastases observed in carcinoma, say, of the mamma. This is to a large extent the case, but it does not justify the statement that the earlier view of blood-spread must be abandoned and that "the spread, as was shown by workers at the Middlesex Hospital, is invariably by the lymphatics"[3].

The present case illustrates the following points: (1) Extensive involvement of the distal bones of the limbs; (2) Departure from the rule that the liability of a bone to cancer metastases is increased by its mere proximity to the primary growth; (3) The fracture of the left femur high up in the upper third of the shaft and not lower down where compact bone is thinner; (4) The fracture of the left humerus below the insertion of the deltoid muscle; (5) The freedom of the sternum from carcinomatous involvement, which is unusual, as it often is involved in cases of direct or of lymphatic spread; (6) Very few skin nodules, although the osseous-spread was very diffuse.

Perusal and careful consideration of the pathological report point to this case being a combination of lymph- and blood-spread from the primary tumour.

The *lymph-spread* is manifest in the skin nodules, the enlarged axillary glands, the involvement of the pleuræ, and in the osseous invasion of the vertebræ, ribs, clavicle, and skull. In the clavicle the cancer had spread by the costocoracoid membrane, and in the skull by the cervical fascia, which gave rise to numerous nodules lying outside the cranial bones under the periosteum forming bosses and eroding the bones. There were also nodules found under the dura mater which had travelled probably by the fascia between the

sutures, and thus were lymphatic in their origin. Fascial spread also occurred in the case of the vertebræ and the ribs.

The *blood-spread* is shown in connection with all the long bones of all the limbs, with the liver, lungs, and kidneys. In the case of the left humerus, the infection was apparently through the marrow, possibly by the nutrient artery, for careful examination of the various muscles, tendons, and fasciæ going to the humerus furnished no evidence of direct spread of cancer cells by these routes. The only place where any growth was found in the muscles and fascia was around the seat of fracture, and this condition is easily understood. An examination of the right unfractured humerus gave similar results, although the medulla was full of tumour tissue. The state of matters in the left femur was also corroborative of blood-spread. In the fractured bone the whole marrow was more or less replaced by yellowish-white tumour tissue, with some red marrow still seen here and there towards the centre of the shaft. In this bone, again, there was no evidence of tumour growth at the point of muscle insertions, nor of spread along the tendons, fascia, etc. The only thing noticed was slight infection of the vasti muscles at the seat of fracture. The conditions just enumerated were those also present in the right unfractured femur. In the case of the radii and ulnæ, the shafts showed distinct deposits (*Fig.* 303), as also in the tibiæ and fibulæ. In the latter, nothing was found along the attachments to their proximal ends. Confirmatory evidence of blood-spread is furnished by the nodules present in the liver and kidneys. In the case of the liver, the nodules were, as the post-mortem report states, scattered through the organ and were both in its substance and near the surface. The nodules in the centre were larger than the surface ones, a fact that would suggest that they had arrived through the blood-stream, especially as some of them showed definite relation to portal tracts. The infection of the kidneys is in keeping with blood-infection, for some of the numerous nodules dotted throughout them manifested a definite relation to the blood-vessels, while others were just under the capsule with a definite layer of renal tissue between them and the capsule. Lastly, the important link in all blood-infection is furnished by the condition of the lungs, which were studded throughout with small nodules of growth, whitish, and the size of a small pea, and were most probably spread by the blood-current, as there was no evidence that they had travelled along the bronchi.

There are other points in this case to which attention might be drawn, but sufficient has been said in support of the contention that it is clearly confirmatory of the embolic theory of metastases, and demonstrates that both it and lymphatic infection may go on side by side in the same patient, thus indicating the need for some modification of the dictum laid down by the Middlesex Hospital School, that "the spread of cancer is invariably by the lymphatics". Further, it is in no way confirmatory, but rather contradictory, of the contention very strongly emphasized by Handley that there is a relation between skin-infection and bone-infection—in other words, that there exists a resemblance in extent between the areas liable to cutaneous nodules and those liable to metastases in bone. The present case is also quite in keeping with the conclusions come to by Piney[4] in his valuable paper in this journal, where he brings forward very strong proof that cancerous

metastatic deposits in bones are due to arterial or capillary embolism, in the shape of evidence that vascular channels in the bone-marrow contained both red corpuscles and epithelial cells, side by side, and that his investigations render it an open question as to the existence of lymphatic channels in bone-marrow.

SUMMARY.

1. Carcinoma of right mamma with *lymph-spread* to skin, glands, pleuræ, clavicle, skull, and vertebræ; and with *blood-spread* to all the long bones of all the limbs, and to the liver, lungs, and kidneys, so that the case is partly lymphatic- and partly blood-spread.

2. The blood condition is of interest, showing diminution of red cells with marked increase of leucocytes, mostly of the polymorph type.

3. Skiagrams show that the cancerous foci are not more frequent in the tendinous or fascial insertions, a point well illustrated in the phalanges of the hand, where the tendons are attached at the base, while many foci are more numerous at the apices. They also demonstrate the presence in the radii, ulnæ, tibiæ, and fibulæ of large nodules and masses of tumour tissue, chiefly in the marrow cavity.

REFERENCES.

[1] LAZARUS-BARLOW, W. S., *Practitioner*, 1914, March.
[2] PAGET, STEPHEN, "Distribution of Secondary Growths in Cancer of the Breast", *Lancet*, 1889, March 23.
[3] *Edinburgh Review*, 1924, July, 108.
[4] PINEY, ALFRED, "Carcinoma of the Bone-marrow", *Brit. Jour. Surg.*, 1922, x, 235.

AN INQUIRY INTO THE CAUSATION OF POST-OPERATIVE PNEUMONIA.

By HENRY FEATHERSTONE, Birmingham.

Since the dawn of civilization, knowledge of the cause of disease has been the principal aim of medical research. But how seldom have men reached the goal! The specific organisms which, as we believe, are responsible for some of the infectious fevers, have been discovered, yet we are still ignorant as to the mechanism which, as the disease appears in different individuals, alters its character and may prevent its development.

The subject under discussion is one of great interest to surgeons and to anæsthetists, for infective diseases of the lung are a serious menace to many patients who have undergone successful operation. To the physician and to the pathologist, the study should repay attention, for the malady begins, runs its course, and ends while the patient is under complete medical control. Theoretically, we should be able to analyse each possible causal factor, and, from our conclusions, draw up an explanation which will enable us to check the scourge in the future.

How far the present essay has succeeded in carrying out this principle is for the judgement of others, but I trust that the time devoted to its perusal may be not ill-spent.

INTRODUCTION.

Material.—The following material is employed in the discussion :—

1. Pertinent literature from Germany, France, Spain, the United States, Canada, and this country, from 1895 till the present day. In the course of the past twenty-five years, these considerable papers have been collected by my senior colleague, Dr. W. J. McCardie, and I have to offer him my grateful thanks for permission to use them.

2. An analysis of the results in one thousand consecutive medical and surgical autopsies, performed between May, 1920, and May, 1922, at the General Hospital, Birmingham, by Professor Shaw-Dunn and his assistants. A general comparison is made with the findings in the post-mortem room at the Queen's Hospital, Birmingham, during the same period. For permission to use this evidence I must thank Professor Shaw-Dunn and Dr. R. G. Abrahams.

3. A comparison of the incidence of pneumonia after operations on the stomach and after hysterectomy, in the series of these procedures which occurred at the General Hospital, between May, 1920, and May, 1922.

4. Records of pneumonia occurring in the surgical wards at the General Hospital from March, 1922, to March, 1924.

5. My own observations as anæsthetist at the General Hospital, the Ear and Throat Hospital, and the Children's Hospital.

Incidence.—In 1897, Dr. Silk[1] referred to the rarity of post-operative pneumonia, and recorded that during 1894 and 1895, 5000 anæsthetic cases were followed by pneumonia on only 13 occasions, of which no fewer than 8 succeeded operations on the tongue or jaw. Prescott, of Boston, had noticed 3 pneumonias after 40,000 administrations. During the next year or two, Stanley Barling,[2] Pridgin Teale,[3] Rumboll,[4] and Miss Aldrich-Blake[5] related similar experiences. Dudley Buxton,[6] Carter Braine, and Silk, in 1900,

Table I.—Incidence, Mortality, and Morbidity of Post-operative Pneumonia.

Author	Operations	Pneumonias		Mortality of Morbidity	Upper Abdominal Operations	Pneumonias		Mortality of Morbidity
		Number	Per cent	Per cent		Number	Per cent	Per cent
1898. Schultz.. ..	5724	27	0·47	70	—	—	—	—
1898. Anders ..	57,842	46	0·07	—	—	—	—	—
1897. Silk	5000	13	0·26	—	—	—	—	—
1898. Barling.. ..	2000	1	0·05	—	—	—	—	—
1900. Rumboll ..	1500	1	0·075	—	—	—	—	—
1900. Aldrich-Blake ..	1250	3	0·24	—	—	—	—	—
1900. Buxton ..	4914	17	0·34	—	—	—	—	—
1900. Julliard ..	4000	0	—	—	—	—	—	—
1900. Carter Braine ..	4380	1	0·23	—	—	—	—	—
1900. Silk	900	1	0·11	—	—	—	—	—
1901. Mickulicz and Henle ..	1787	143	8·0	46	1787	143	8·0	46
1902. Crouch and Corner	3000	10	0·3	10	—	—	—	—
1904. Bibergeil ..	3909	283	7·2	—	3909	283	7·2	—
1904. Gebele	1196	77	6·43	—	1196	77	6·43	—
1904. Albanus ..	1140	53	4·64	—	1140	53	4·64	—
1905. Czerny ..	1302	52	4·0	—	1302	52	4·0	—
1905. Kausch ..	1880	45	2·24	—	1880	45	2·24	—
1905. Kronlein ..	1400	8	0·57	—	1400	8	0·57	—
1906. Lawen	9755	180	1·84	65	—	—	—	—
1906. Armstrong ..	2500	55	2·2	64	—	—	—	—
1908. Wight	9000	31	0·34	19	—	—	—	—
1909. Risley	1000	11	1·11	37	1000	11	1·1	37
1913. Beckman ..	6825	27	0·4	—	—	—	—	—
1917. Cutler and Morton	3490	65	1·86	—	—	—	—	—
1917. Vedin	1413	15	1·06	46	—	—	—	—
1918. Whipple ..	3719	97	2·6	—	—	—	—	—
1919. Cleveland ..	1940	65	3·3	—	—	—	—	—
1922. Herb	1534	76	—	49	1534	19	1·28	—
1921. Mandl	1585	—	8·5	—	1379	—	14·00	—
1922. Smith	221	—	—	—	221	—	7·5	—
1922. Elwyn ..	2932	81	2·76	—	1080	—	6·2	—

recorded 17 pneumonias after 4914 administrations, 1 after 4380, and 1 after 900 respectively. J. M. Anders,[7] of Philadelphia, had seen 1 pneumonia after 300 ether anæsthetics. During this time, quoting from the British and American figures, pneumonia occurred in 41 cases after 60,244 administrations, that is to say, in 0·068 per cent. Silk was justified in describing post-operative pneumonia as rare.

By 1906, however, the results were not so satisfactory. Schultz,[8] Läwen,[9] and Le Dentu[10] quoted 28,093 operations, complicated by pneumonia on 868 occasions, i.e., 3·09 per cent. It is significant that many of these operations

were laparotomies, while the earlier series did not include so many operations on the abdomen.

Armstrong,[11] Wight,[12] Beckman,[13] McKesson,[14] Cutler and Hunt,[15] Vedin,[16] Whipple,[17] Henle,[18] McKesson,[19] Cleveland,[20] Isabella Hert,[21] Mandl,[22] Homans,[23] Muller,[24] Smith,[25] Elwyn,[26] and other writers during the last fifteen years, have reported series of cases in which the morbidity varies from 0·5 to 8 per cent.

The series of records shown in *Table I* indicates, where noted, an exceedingly high death-rate; thus, from 40 to 60 per cent of the patients who developed pneumonia died. It appears likely that, in most surgical clinics, lesser attacks of pneumonia escape recognition; only the more severe cases attract attention, and of these a very large proportion prove fatal. My own experience at a large general hospital of 400 beds has demonstrated how difficult it is to obtain reliable information concerning the incidence of this disease. A system of notification of all suspicious 'chest' cases occurring after operation has been in force for two years, but owing to the frequent changes of resident officers, and the pressure of other work, mild or abortive attacks of pneumonia often escape observation.

Table II.—Incidence of Pneumonia found Post Mortem in Medical and Surgical Cases.

	Description of Cases	At Queen's Hospital	At the General Hospital
Medical	Number of medical autopsies (excluding primary pneumonias)	283	350
	Number of secondary pneumonias occurring in the above	97	89
	Percentage of deaths in pneumonia cases	34·27	25·42
Surgical	Number of autopsies on patients who died shortly after operations under local or general anæsthesia	178	385
	Number of pneumonias (excluding empyema cases)	75	138
	Percentage of pneumonias to deaths	42·13	35·84

Table II demonstrates that pneumonia is a frequent complication in any killing disease whether an operation was performed or not.

Hospitals which deal with the less severe surgical operations report that post-operative pneumonia is extremely rare. Dr. W. R. Jordan,[27] who in the course of thirty years administered chloroform on 11,200 occasions, at the Birmingham Royal Orthopædic and Spinal Hospital, met with but one case of this complication. In my work as anæsthetist at the Birmingham Ear and Throat Hospital, I have only heard of three or four mild cases of pneumonia during a period of four years. But on the other hand, at the General Hospital, from May, 1920, to May, 1922, 222 consecutive severe operations on the stomach (that is to say, gastrectomy, gastrojejunostomy,

or suture of perforated ulcer) were complicated by pneumonia in 24 cases, i.e., 10·81 per cent.

The foregoing records illustrate that pneumonia may occur often or seldom in different series, and that our analysis must be wide and searching if we are to find the cause.

Course and Varieties.—The first symptoms may occur a few hours after operation or not until after many days,[31] but the onset is rarely earlier than twenty-four hours or later than five days after operation. If more than a week has elapsed before pneumonia sets in, we usually find some other cause, such as progressive failure, and both operation and anæsthetic may justly be exonerated.

The subsequent course, naturally, differs with the variety of the disease, but most surgeons, I find, recognize two main types. The first we may call 'the abortive type', which corresponds to the 'pneumonitis' described by Whipple.[17] Never by itself fatal, its sudden onset is characterized by absence of chill or rigor, by sharp rise of temperature, moderate cough, and some pleuritic pain. The temperature falls by lysis after forty-eight hours. During the first few hours radiography shows a shadow in the lung " often wedge-shaped, and usually in the lower lobes ". Over the affected area there is dullness on percussion, and the breath sounds are diminished. Later, tubular or even bronchial breathing may be heard. In 11 out of 97 cases, radiographic examination showed a shadow before the physical signs of consolidation were elicited. Mason[30] thinks these shadows begin at the periphery and extend to the hilum. He suggests that bronchial breath sounds are not heard until the shadow extends from the periphery to the hilum. Rusty sputum is rare, but yellow mucus is expectorated, from which the *Group IV* pneumococcus may be grown both before and after operation.

In his account, Whipple does not mention any displacement of the heart, but in many respects his description tallies with that which Pasteur[29] gives of 'massive collapse' of the lung. According to Pasteur, there are two well-defined clinical types of massive collapse, one acute, the other latent. The acute type may present a sudden initial dyspnœa resembling pulmonary embolism, or, more commonly, a rapid but less dramatic onset suggesting pneumonia. In the latent type " there may be a complete absence of symptoms, although the physical signs are well developed ". Undoubtedly these cases often escape attention. Pasteur, however, refers to " a sudden onset ", " deep-seated pain in the lower part of the chest ", viscid mucous expectoration which is not blood-stained, dullness over affected base, diminished breath sounds or tubular breathing, loss of vocal fremitus, and a sharp rise in temperature to 103°. He tells us that the symptoms rapidly subside in the absence of the development of secondary bronchopneumonia, but that this is quite likely to occur. He emphasizes paresis of the diaphragm, often unilateral, imperfect movement of the affected side of the chest, and over-action of the opposite side. Of primary importance, the heart is dislocated towards the affected side. Displacement of the heart he regards as pathognomonic. By radiography, before and after operation, Pasteur has satisfied himself that paresis of one half of the diaphragm is a frequent result of abdominal and other operations, and this often passes off unobserved. Further space will be devoted to the consideration of the mechanism of these conditions.

The second principal variety of pneumonia is extremely grave. I recognize a type of spare middle-aged man, often with emphysema of the lung and very little costal movement on respiration, who, though in fair general health, is poorly nourished as the result of gastric or duodenal ulcer. In the course of an hour, a severe operation is performed on his stomach, and he recovers consciousness uneventfully, although rather pale and weak. Twenty-four hours or more after operation, the colour becomes cyanotic, the pulse and temperature rise, the lungs give evidence of patches of consolidation or at least of areas of collapse, and only too frequently death ensues on the fourth, fifth, or sixth day after operation. In the series at the General Hospital, after 106 'clean' stomach operations on men of this class, taken consecutively, pneumonia set in in 21, and of these 13 died. The morbidity was 19·81 per cent, and the mortality was 61·9 per cent of the morbidity.

In addition to these two main kinds, we see examples of terminal lobular pneumonia in exhausted patients, suppurative embolic pneumonia in cases of severe sepsis, hypostatic pneumonia, suppurative aspiration pneumonia after operations on the tongue or jaw, pneumonia lit up in lungs which recently have been the seat of inflammatory disease, but I believe lobar pneumonia to be extremely uncommon. In 138 autopsies on post-operative pneumonia cases at the General Hospital, there were 136 instances of the lobular variety and only 2 with definite lobar distribution.

Bacteriology.—This branch of the subject bristles with difficulties, and much work remains to be done.

In cases of definite blood infection from a septic focus elsewhere, e.g., osteomyelitis of bone, usually streptococci or staphylococci of the same character as those in the primary abscess are discovered in the lung. The cultures are not always pure, and quite often the guilty microbe cannot be demonstrated with certainty.

From cases of pneumonia due to aspiration of particles of vomit or mouth contents, many varieties of germs may be grown in profusion, and the formation of multiple abscesses is found to be of common occurrence.

Whipple,[17] whose research on this subject is by far the most important of those which have been published, found the pneumococcus in nearly all air-borne infections. Neufeld and Handel[33] and, later, workers at the Rockefeller Institute showed that pneumococci may be divided into strains by the reactions of animals immunized against pneumococci obtained from various sources. Four groups have been distinguished. Pneumococci labelled *Groups I, II, III* are parasitic and are found only in the throats of patients who have been exposed to infection, while *Group IV* pneumococci are saprophytic and are present in about 60 per cent of normal persons. Whipple injected the pre-operative sputum into a mouse; if the pneumococcus was found, it was cultured and kept. If pneumonia occurred after operation, the culture from the pre-operative and the culture from the post-operative sputum were tested for *Group IV* pneumococcus. If this variety was found, specimens of blood were taken at three-day intervals and serum agglutination tests were employed with both the above sets of cultures. Whipple " concludes that the majority of post-operative pneumonias have pneumococci of *Group IV* in the sputum; especially is this true in the milder cases, in which the pneumonia develops in

otherwise healthy individuals, and runs a short atypical course ".[17] Pneumococci of *Groups I*, *II*, and *III* are the causal organisms in severe medical pneumonia and are often found in post-operative pneumonia occurring in epidemics.

Wollstein and Meltzer[34] found that experimental intrabronchial insufflation of non-virulent pneumococci in dogs caused an exudation into the alveoli which did not affect the frame-work and which usually was absorbed rapidly, the exudate being very poor in fibrin. Perhaps this is an experimental reproduction of the transient pneumonia which follows invasion of the lung by *Group IV* pneumococcus during or after surgical operations.

Important as this work is, very little has been said authoritatively by other investigators, either in confirmation or in contradiction. During a period of several months, I took post-nasal swabs of patients about to be anæsthetized for severe abdominal operations. Direct smears showed a great variety of organisms, including the pneumococcus, but pneumonia did not occur in the series, so that the research was fruitless. In a more recent case—a man, 35 years old—owing to the presence of pharyngeal catarrh operation was deferred for ten days. At the end of this time, the temperature having been normal for six days, upper abdominal laparotomy and appendicectomy were performed. A mild attack of 'bronchopneumonia' commenced on the second day following operation, and all signs disappeared by the seventh day. Before operation pneumococci were found on a post-nasal swab, and after operation a copious growth of pneumococcus was prepared from the sputum. We were unable to ascertain the particular strain of pneumococcus. Probably it belonged to *Group IV*, but cultures of the pneumococcus of known variety were unobtainable at that moment.

To sum up, Dr. Whipple's views are probably true, and, so far as I know, they are borne out by the experience of other workers. A first-rate knowledge of bacteriology, the necessary certificates, much spare time, and ample funds are essential to the pursuit of this research. Unfortunately, hospital bacteriologists and hospital anæsthetists do not possess all these qualifications.

THE POSSIBLE CAUSES.

For many years, in the absence of some obvious cause such as septicæmia, pneumonia which followed operations under general anæsthesia was attributed to the anæsthetic agent. In the following pages, I shall deal with the subject on the widest possible basis, endeavouring to examine and to assess each unaccustomed influence which is brought to bear on the patient at operation.

I think the various items may be discussed conveniently under the following general headings: (1) *The anæsthetic agent.* (2) *The methods of administration of the anæsthetic.* (3) *The operation itself.* (4) *The patient's general condition.* (5) *General systemic disorders affecting metabolism and organs of defence.* (6) *The presence of pulmonary disease prior to operation.* (7) *The patient's respiratory system.* (8) *The process by which the lung becomes infected.*

1. THE ANÆSTHETIC AGENT.

Usually one of three agents is employed to give prolonged general anæsthesia—ether, chloroform, or nitrous oxide and oxygen. I shall review these *seriatim*, together with some remarks on the results obtained with local analgesia.

Ether.—There is a considerable weight of evidence to show that ether is a powerful irritant to the mucous membrane of the respiratory passages. That impure ether, such as the methylated ether of commerce, may be the starting-point of an attack of pneumonia, appears very likely. Dr. Lamb[36] has quoted an outbreak of pneumonia which followed the use of commercial ether supplied by a hospital chemist for reasons of economy. Recently, Professor Storm van Leeuwen[65] demonstrated in some specimens of ether the presence of highly irritant substances which may be extremely dangerous, even in small quantities.

Hölscher,[35] in his classical paper, describes the action of ether vapour on the various regions of the respiratory system with which it comes in contact. In the nose and mouth, profuse watery secretion and salivation are excited, but in the trachea, ether acts as a local anæsthetic, and, while paralysing the cilia of the tracheal epithelium, ether insufflation through a tracheotomy tube, or by an intratracheal catheter, fails to produce local secretion, and there is no salivation. The large bronchi do not excrete fluid, but the alveolar epithelium may secrete vigorously. It is found that the amount of secretion varies with individuals and with different species. Some patients may be anæsthetized with pure ether without the usual pre-operative dose of atropine, yet no secretion or salivation will result, while others rapidly become 'water-logged'. Rabbits are readily anæsthetized with ether, and remain 'dry'; but cats produce an abundant secretion. In man, after an operation, sometimes the ether is not quickly excreted, the effect of the atropine wears off, and plentiful frothy secretion, together with a certain amount of œdema of the lung, may result. In these cases a further dose of atropine has an extremely beneficial action.

Large doses of ether vapour undoubtedly lead to petechial hæmorrhages in the lungs of rabbits, as I can testify from my own observations. Poppert[37] made dogs inhale strong ether vapour and rapidly produced hæmorrhage and œdema in the lungs. When the administration was arrested the œdema quickly subsided; this has suggested that the ill-effects were the direct result of irritation alone, and that, in these experiments, the hæmorrhage and œdema was not inflammation produced by infection of the irritated tissues. Chapman[38] poured liquid ether on a frog's web, thereby causing arrest of the circulation, and rupture of the vessels. In other experiments on rabbits this observer found that: (*a*) Etherization caused congestion of the alveoli, while with very concentrated vapour intra-alveolar hæmorrhages ensued. (*b*) Cultures of pneumococci were blown into the lungs during ether narcosis; this was repeated daily for three days, and bronchopneumonia followed. (In my opinion this experiment was too severe for us to formulate any reliable conclusions.) (*c*) Suffocation alone produced hæmorrhage of the lung.

When we compare these results with those of clinical practice, we find

much that is conflicting. Hæmoptysis after ether anæsthesia is almost unknown, and many workers have commented on the infrequency of blood in the sputum of post-operative pneumonia cases. Further, if ether were so irritating as an anæsthetic, we should expect laryngitis and tracheitis as frequent sequelæ to etherisation, but in practice these complications are almost unknown.

Anders,[7] Drummond,[39] Gerulanos,[40] Dupley and Hallion,[41] Crouch and Corner,[42] Wight,[12] and others have blamed ether as the most potent causal factor. In particular, Crouch and Corner[42] kept careful records of 2400 administrations of ether at St. Thomas's Hospital. Of these, 10 developed pyrexia and respiratory trouble within twenty-four hours, 1 of whom died. Not one patient of 600 who received chloroform showed this early respiratory disease, which is the more surprising because many of them had undergone mouth and tongue operations, which are common precursors of lung trouble.

On the other hand, Hölscher, Reamy,[43] Le Dentu, Silk, Cutler and Morton, Whipple, Elwyn,[26] and many others do not blame ether as the most potent factor in the production of bronchopneumonia after operations. At the German Surgical Congress held in 1905, of nine surgeons, three blamed ether, one exonerated ether, and five did not deem ether or chloroform very guilty. Silk[6] has said that post-operative pneumonia was extremely common before the days of anæsthetics and antiseptics; indeed, he has known of ether being administered to cases of pneumonia without affecting the course of the disease. I can confirm this from personal experience. Many of us can recall cases of subacute bronchitis clearing up rapidly after etherization.

Now, many anæsthetists consider ether should not be given in too great concentration. Mrs. Dickenson Berry,[36] who possesses unrivalled experience with light ether anæsthesia for thyroidectomy, records over one thousand administrations of ether with only one pneumonia. My colleague, Dr. Hassall, thinks bronchitis, or even pneumonia, may be expected in alcoholic persons, who struggle during induction and inhale massive doses of nearly pure ether vapour. Certainly, depth of ether anæsthesia is not to be blamed, for I have records of 1179 operations for nose and throat affections performed by Mr. Musgrave Woodman under prolonged deep ether narcosis, followed only in 1 case by pneumonia (after diathermy of a malignant ulcer in the mouth). But deep anæsthesia should not be attained with concentrated vapour; free respiration and uniform inhalations of a vapour of regular strength (say, 20 per cent) rapidly lead to deep narcosis with a good circulation, effortless respiration, and little or no irritation of the lung tissue. In this connection I may quote Dr. Geoffrey Marshall's[45] results with cold and warm ether vapours (in France). Fifty-four per cent of abdominal cases developed bronchitis or pneumonia after 'open ether', while with warmed ether vapour chest complications ensued in only 14·7 per cent. Dr. Shipway, one of the pioneers of the 'warmed ether vapour technique', has obtained similar results. It seems reasonable to suppose that the rapid evaporation of large quantities of ether on a soaked open mask causes great cooling of the apparatus and the formation of a fog of ether droplets, instead of the warm, non-irritating gas produced by the warmed ether apparatus (cf. the animal experiments quoted above).

Many writers very wisely draw attention to the exhaustion produced by

the prolonged administration of ether. I think this may be reduced by careful attention to free respiration, warmth, etc.

We may note that Graham[46] showed that ether checks phagocytosis in the body, and also in blood treated with ether *in vacuo*. He found that fats and lipoids reduced this inhibitory action of ether, and that a subcutaneous injection of lecithin, or a rectal administration of olive oil, rapidly restored the opsonic index count to normal. On the other hand, Dr. Flemming,[47] in a few estimations, always found that after ether anæsthesia the opsonic index count was raised.

I now turn to the consideration of certain results obtained at the General Hospital, from May, 1920, to May, 1922. During this period, 222 operations were performed on the stomach. The nature of the operations was gastro-enterostomy, partial gastrectomy, or suture of perforated ulcer.

Ether was given in	158 cases;	pneumonia occurred in	15 =	9·43 per cent.
Chloroform* was given in	64 cases;	„ „ „	9 =	14·06 „

The post-mortem records for this period give interesting information. In 385 autopsies on patients who had died within ten days of operation, pneumonia was found in 138.

Of 138 pneumonia subjects :—

Ether was used in	67 =	48 per cent.
Chloroform was used in	53 =	38 „
Gas and oxygen was used in	6 =	4 „
Local anæsthesia was used in	12 =	8·7 „

Of 385 surgical autopsies :—

Ether was used in	203 =	52 per cent.
Chloroform was used in	110 =	28 „
Gas and oxygen was used in	37 =	9 „
Local anæsthesia was used in	35 =	9 „

Thus, speaking generally, we may conclude that, although ether had been employed in *more* than half of these fatal surgical cases, ether had been the anæsthetic in *less* than half of the pneumonia subjects. On the other hand, chloroform had been used in *less* than one-third of the fatal subjects, but chloroform was the anæsthetic in *more* than one-third of the pneumonias. Probably the more frequent occurrence of chloroform as a precursor to pneumonia was due to the expectation of lung trouble in these subjects when the anæsthetic agent was being selected. (It may be noted that local anæsthesia had been employed in about 9 per cent of the total surgical autopsies, and that it had been used in the same percentage of those in whom pneumonia had occurred.)

Finally, we are forced to the conclusion that relatively pure ether when administered with reasonable care is not an important cause of pneumonia, and that, most certainly, the routine use of chloroform—a practice still advocated by some surgeons—would not rid us of this grave complication.

---

*Under the heading of 'chloroform' is included E_2C_1 mixture, of which the quantity of ether inhaled by the patient is negligible. It will be observed that pneumonia occurred more commonly after chloroform.

Chloroform.—In his experiments with chloroform on twenty-two rabbits, Von Lichtenburg[48] found grave alteration in the lung tissue, including swelling and disintegration of the epithelial cells in the alveoli. This led to broncho-pneumonia in several instances. Dr. Flemming[47] has demonstrated the action of ether and chloroform on *Chlamydonas* (a free-swimming vegetable cell), on free-swimming protozoa (such as *Paramæcium*), and on the cilia of the buccal mucous membrane of frogs. In each kind of cell he found that chloroform, in quite weak dilution, arrested movement, but that a stronger concentration of ether was required to produce paralysis. Furthermore, in all experiments, the etherized cells recovered much more quickly than those under chloroform; indeed, in most cases the chloroform had killed the cells.

Now, Poppert, in his experiments with chloroform on dogs, produced less œdema and smaller hæmorrhages than had been seen after the administration of ether. However, we may conclude that in some patients chloroform acts destructively on the respiratory epithelium and may lead to bronchitis or to pneumonia.

The published clinical results are contradictory. Le Dentu,[10] in a review of 800,000 cases of general anæsthesia, found one death after operation under chloroform anæsthesia in every 2500 administrations, and he considered chloroform to be quite a powerful lung irritant. He states that Richet and Championnière found that chloroform was the anæsthetic in the majority of their post-operative pneumonias. Campiche,[49] after 511 ether cases, recorded lung trouble in 3 per cent, but after 205 chloroform administrations lung trouble was observed in 5 per cent.

Riley,[50] writing from Cuba, states that chloroform is his routine anæsthetic agent, and that he never sees pneumonia after operation. Judging from our experience of pneumonia after local anæsthesia and following injuries, we wonder if the infecting organisms are as rife in Cuba as in this country. Again, in the considerable experience of Dr. McCardie, pneumonia has been an extremely rare complication of chloroform anæsthesia. Dr. Jordan, too, states that he has given chloroform to 11,200 consecutive patients for ortho-pædic operations, with only 1 pneumonia.

That pneumonia does occur only too frequently after chloroform anæsthesia is demonstrated by the findings at autopsy and the records of gastric operations mentioned above.

Owing to the unquestionably poisonous effect of chloroform on the heart, mixtures of ether and chloroform have been employed very generally. Poppert, in a series of 812 administrations of a mixture of 5 parts of ether and 1 of chloroform, had to record 5 deaths from pneumonia. It must be noted that all these fatalities occurred after operations on the stomach or gall-bladder, that is to say, after upper abdominal trauma.

In many hospitals it is the custom to employ a mixture of 2 parts of ether to 1 of chloroform, i.e., E_2C_1. For all practical purposes this gives chloroform anæsthesia; first, because it must be evaporated on a thin layer of gauze or lint, which permits the small amount of volatile ether to disperse rapidly, so that little or no ether reaches the lungs of the patient; and secondly, because, even were the ether and chloroform to be inhaled in the proportions of 2 : 1, ether only possesses one-eighth of the narcotizing power of chloroform,

so that anæsthesia will still be due to chloroform. (It may be noticed that Rodman, Thompson, Kemp, and others deprecate the use of these mixtures.)

It appears, then, that although there is some evidence that chloroform may play a part in the production of pneumonia, some other more powerful factor is nearly always present.

Nitrous Oxide and Oxygen.—Most authorities agree that lung complications following the use of gas and oxygen would be extremely rare if we did not select it as the routine anæsthetic for cases in which lung trouble is expected. For example, in my post-mortem records, 6 of the subjects had each undergone two operations, the last of which was of a desperate nature. In each of them, a second operation had been performed under gas and oxygen anæsthesia. No fewer than 5 of the 6 showed consolidation of the lungs at autopsy. In this post-mortem series, gas and oxygen had been employed for operations on 37 subjects; 6 had developed pneumonia, and of these, 5 pneumonias followed desperate ante-mortem operations. Of the anæsthetics known to us, nitrous oxide is the least irritating to the respiratory system and the most harmless when absorbed. It is unfortunate that we cannot employ gas and oxygen alone for operations on the abdomen, but the addition of a little ether is a great help.

Mr. H. J. Patterson[36] has shown that post-operative rise of temperature after gas and oxygen is less than that following ether or chloroform. Frequently it is my practice, in severe upper abdominal operations, to induce deep ether anæsthesia for laparotomy and exploration of the interior of the abdomen. During the suturing and quiet manipulations which follow, gas and oxygen, under positive pressure, is employed, and later on a little ether is added during the closing of the abdomen. Nevertheless, I have to record during the last year, after the use of this method, one case of mild pneumonia and one which proved fatal. The occurrence of pneumonia, however, was due to other factors.

Local Anæsthetics, including Spinal Analgesia.—In 1901 Henle[51] found pneumonia to be even more frequent after local analgesia than after general anæsthesia. With this conclusion Goebel,[52] Läwen,[9] Hendry,[53] Mickulicz,[54] and Griffin[55] agree. Gastrostomy, whether performed under local or under general anæsthesia, is a frequent precursor of pneumonia. Goebel mentions 4 cases of bronchopneumonia after this operation, while Läwen, in 1955 operations, found pneumonia in 36 per cent of the gastrostomies. Hendry quotes Gottstein's 114 laparotomies under cocaine, 16 of which were followed by lung œdema and 'hypostatic' pneumonia.

Mandl[22] records 1283 operations under general anæsthesia with lung complications in 4·66 per cent, and 910 operations with local analgesia followed by respiratory trouble in 4·39 per cent.

On many occasions, in his experience of 8000 cases, Rood[56] has seen bronchitis and pneumonia follow spinal analgesia and local analgesia. In a series of 100 administrations of spinal analgesia, of which the most careful records were kept, the present writer[57] saw 2 cases of severe broncho-pneumonia, both of which recovered. One was a woman suffering from loss of blood and diffuse peritoneal shock due to rupture of a tubal gestation. She had a cold at operation, so spinal analgesia was induced. Next day severe

capillary bronchitis had developed, and during the following week widespread bronchopneumonia was present.

Cæsarean hysterectomy was performed on the second patient for severe toxic intra-uterine hæmorrhage at the ninth month of pregnancy. She was pale and bloodless before operation and very shocked afterwards. Within forty-eight hours of the operation, bronchopneumonia was present throughout the left lung, but, after pyrexia which lasted fourteen days, she made a complete recovery.

I do not advocate *high* spinal analgesia, so that, with one exception, I am unable to discuss the effect of paralysis of the intercostal muscles from this cause in the production of disorders of the lungs.

In an exceedingly stout woman, however, owing to the exaggerated recession of the lumbar curve, it was necessary to inject the tropacocaine solution into the lower dorsal region where the vertebral column approached most closely to the skin. High analgesia and loss of motor function to the fifth intercostal space was the result. The patient, without any increase in pulse-rate or respiration, became very cyanosed. Dr. Jonathan Meakins[58] points out that in spinal paraplegia involving the lower intercostal muscles carbon dioxide accumulates in the blood, and oxygenation is deficient. It is well known that lesions of the dorsal region of the spinal cord easily lead to bronchopneumonia. For other reasons, high spinal paresis is, perhaps, to be deprecated.

I am informed that some surgeons when performing gastrectomy are obtaining good results from paravertebral analgesia involving the eighth, ninth, and tenth dorsal nerves, but it is too early for us to form conclusions.

2. THE METHODS OF ADMINISTRATION OF THE ANÆSTHETIC.

When considering the effect of ether on the respiratory system, I enumerated certain essential points to be watched in the administration. In one respect the inexpert anæsthetist will spare his patient, for he will not give a very strong vapour lest an overdose occur. Thus, although satisfactory anæsthesia may not be attained in every case, great irritation of the respiratory epithelium from concentrated vapour will be avoided.

Open Methods and Closed Methods.—It is evident that the ideal method of administration will provide completely vapourized anæsthetic material of correct uniform strength. With this end in view, the late Joseph Clover some fifty years ago introduced his well-known closed ether inhaler. At one time it was suggested that this apparatus might be infected by patient A, and that the next patient, B, would be infected with the disease of patient A by the inhaler, but we should remember that ether is a mild antiseptic, and that a gauze mask soaked with ether, or a Clover's bag charged with strong ether vapour, is comparatively free from organisms.

Offergeld[10] stated that animals anæsthetized by the closed method sometimes developed bronchopneumonia, whereas he had never seen bronchopneumonia follow anæsthesia induced by the open method. Wight[12] considered that ether dropped on a few layers of gauze reduced the number of respiratory troubles. Dr. T. L. Hardy,[59] at the Middlesex Hospital, found pneumonia

more frequently after closed methods than following administration on an open gauze mask. Vedin,[16] who preferred simple methods, gave no preliminary atropine, and induced light anæsthesia by means of 'open drop ether.' Nevertheless, in 1915, after 1413 operations, there were 15 cases of pneumonia (1·06 per cent), and of the 15 patients attacked, 7 died.

There are plenty of doughty champions to support the cause of the closed inhaler. Silk said the closed inhaler was not a causal factor in this trouble, and he instanced the absence of lung trouble after administrations of gas. Acapnia, according to Yandell Henderson,[60] often resulted from open ether, and this was complicated by respiratory and circulatory depression, by post-operative vomiting, by intestinal paresis, and by gastric pains; therefore he advised closed methods. Dr. McCardie[61] thinks there was not so much bronchopneumonia in the days when the Clover inhaler was used for routine work. Armstrong, of Montreal, had implicit faith in this method. At the Birmingham Ear and Throat Hospital, where it is the custom to administer ether by the Clover inhaler, post-operative pneumonia is comparatively rare, one or two cases occurring in each year.

The choice of method can play very little part in the causation of the disease which we are considering.

The Intratracheal Method.—Poppert[37] stated that, in dogs, intratracheal insufflation of strong ether vapour caused hypersection, hæmorrhage, and occlusion of the lung. Clearly these results are not applicable to man, because this means of administrating ether approaches more closely to the ideal than any other. A regular supply to the lungs of ether vapour of minimal strength is constantly maintained, with the least possible exertion by the patient. Furthermore, when the intratracheal catheter has been inserted, and regular respiration is established, even in the absence of atropine, there is no salivation.

For operations on the tongue and jaw, anæsthetists agree that the introduction of the intratracheal method has reduced the risk of aspiration pneumonia. My last 20 cases of this class did not suffer from pneumonia.

Of course, the intratracheal technique does not entirely prevent pneumonia. I have seen pneumonia follow intratracheal anæsthesia for mouth operations, for thyroid operations, and for operations on the stomach. On the other hand, this complication has not appeared after intratracheal anæsthesia, in my practice, during a period of twelve months. I attribute the improvement to (*a*) chance, and to (*b*) permitting the cough reflex to return before releasing positive pressure by withdrawing the catheter. In this way I have avoided collapse of the trachea or of a bronchus, which might have led to partial or total collapse of the lung. In this connection the following case notes are of interest :—

Case 1.—B. G., male, age 35. Acute enlargement of the thyroid gland, threatening suffocation. Hemithyroidectomy was speedily performed under intratracheal ether, and the catheter was removed immediately after completion of the operation. During the next forty-eight hours he suffered from attacks of cyanosis. Occasional delirium, with pyrexia, accompanied by reduced entry of air, and râles at the right base, ensued. On the seventh day after operation, the pulse-rate fell suddenly from 100 to 80, and the temperature to normal. This patient's expectoration was scanty. In view of his condition it was deemed inadvisable to radiograph the chest, but I am inclined to think that the attacks of cyanosis, the scanty expectoration, and the sudden recovery were signs of collapse of the lung, initiated, in all probability, by collapse of a bronchus or of the trachea after premature removal of the catheter.

Intratracheal insufflation of ether will not prevent the onset of every variety of pneumonia, nor will collapse of the lung always be avoided, but the technique, if wisely employed, will reduce the incidence of post-operative pneumonia.

3. THE OPERATION.

We have now suggested certain principles in the choice of the anæsthetic and in its administration which it is certainly the duty of the anæsthetist to observe very closely, but his responsibility should not end here. Clearly, the anæsthetist should co-operate with the surgeon by keeping watch on the patient's condition before, during, and after operation, by preventing infection, and by sustaining the patient's strength. To the discussion of these topics we will now proceed.

Posture of the Patient during Operation.—The various attitudes in which patients are placed to obtain satisfactory access to the site of operation appear to have some bearing on the causation of lung complications.

When the patient is inclined head downwards in the Trendelenburg position, the risk of aspiration of infected material from the mouth and nose appears to be very small, but inversion of the patient causes the abdominal contents to press down upon the diaphragm, and there is some limitation of expansion in the lower lobes of the lungs. Nevertheless, pneumonia is rarely seen in gynæcological practice; at the General Hospital, only 4 cases of this complication occurred after 400 abdominal sections performed in the Trendelenburg position.

Patients lying horizontally on the back may aspirate material from the pharynx. Through the laryngoscope, on several occasions, I have witnessed the passage of mucus into the larynx. Children bleeding freely after tonsillectomy occasionally draw blood into the trachea, but, fortunately, expectoration expels it after a few moments. Of course, most surgical major operations are performed while the patient is lying in this position, and reference will be made to other points in this connection at a later stage.

The patient who lies face downwards for laminectomy or trephine of the occiput appears to run but little risk of aspiration pneumonia. Costal breathing must be rather restricted, but, in the comparatively few examples of these operations which I have seen, no lung trouble supervened.

I have records of a large series of nose and throat operations performed by Mr. Musgrave Woodman at the General Hospital upon patients seated upright in an operating chair. Dissection of tonsils, extensive nasal operations, excision of the superior maxilla, and intracranial operations, numbering in all over 200, have been done with only 1 case of pneumonia. Perhaps this low incidence may be attributed to the lessened tendency to loss of blood in this position, to careful hæmostasis, and, in the more severe procedures, to the employment of positive pressure intratracheal insufflation.

I believe the Trendelenburg position lessens the risk of aspiration pneumonia, but otherwise the position of the patient during operation is not an important factor in our inquiry.

Site of Operation.—Whatever may be the underlying cause in the production of pneumonia after operations on the abdomen, there is remarkable

agreement among all observers that laparotomy is the commonest forerunner of post-operative consolidation of the lung. Silk, Anders, Le Dentu, Läwen, Armstrong, Ranzi, Beckman, Isabella Herb, Hochenegg (quoted by Mandl), Homans, Cutler and Morton, Poppert, Elwyn, and Whipple have advanced strong evidence in support of this conclusion. Le Dentu quoted many series of abdominal operations recorded by German authors, in which the morbidity rate of pneumonia ranged from 4 per cent to 8 per cent. Läwen's rate was 6·6 per cent of gastric resections. Isabella Herb noted 9 pneumonias after 153 operations on the stomach (5·8 per cent). Poppert administered a mixture of 5 parts of ether with 1 part of chloroform for 812 operations of all kinds; 5 of the patients died, all with pneumonia, in each case after laparotomy. Whipple, in his series of 97 cases of pneumonia following operation, found that in 88 instances the abdomen had been opened. Elwyn's figures are very significant: in a series of 2932 operations, pneumonia occurred after 81 (2·7 per cent); 1080 laparotomies were complicated by 68 pneumonias (6·2 per cent), but 1852 operations on other regions showed a morbidity rate for pneumonia of only 0·7 per cent. Crouch and Corner, in their careful observations at St. Thomas's Hospital, noted 10 lung complications after 2400 administrations of ether, but no case of lung trouble after the employment of chloroform. They concluded that ether was the source of the trouble. Nevertheless, another explanation seems permissible, for 9 of the 10 cases of lung disease followed abdominal operations.

In the General Hospital records of 222 consecutive operations on the stomach, pneumonia appeared in 24 instances (10·8 per cent), 110 hysterectomies were followed by pneumonia in only 2 cases (1·8 per cent). Although, as I have said, the Trendelenburg position must lessen the incidence of pneumonia complicating hysterectomy, yet we may infer that an operation on the upper abdomen is more likely to cause lung trouble than surgical interference with the contents of the pelvis.

At the General Hospital, in 135 instances of post-operative pneumonia found at autopsy, 56 (41·4 per cent) were observed in subjects with recent operation wounds between the ensiform cartilage and the umbilicus. Of the remaining 69, at least 59 had died with large suppurating foci, and well-established septicæmia. Thus, in the absence of widespread sepsis, epigastric operations were the most constant forerunners of pulmonary disease. In 44 post-mortem examinations of epigastric operations at the Queen's Hospital, pneumonia was found in 22 (50 per cent), while in the whole series at this hospital the post-operative incidence was 35 per cent.

Although the mechanism whereby abdominal section predisposes to lung disease will be dealt with when considering the causation of massive collapse, and in other places, perhaps one point may be raised at once. Graham[46] has demonstrated the lowering of the opsonic index after saturation of the blood with ether; he has found that subcutaneous injection of lecithin or rectal administration of olive oil quickly restores the deficiency. Perhaps the shortage of fats in the diet of patients undergoing surgical treatment for stomach trouble may of itself produce a lowering of the opsonic index. During the war, the inhabitants of Europe were unable to obtain a sufficient supply of fat in their dietary, and many believe that the pandemic of

influenza and pneumonia spread and increased in virulence on that account. H. H. Brown[69] suggests that during the performance of gastro-enterostomy a pint of peptonized milk with egg and brandy should be introduced into the jejunum in order to reduce the period of starvation in this method of treatment.

Shock from Operation.—There is no need to discuss at any length the numerous hypotheses which have been elaborated to explain the cause of shock. We may, however, recall Crile and Mummery's earlier view that the vasomotor system was exhausted from prolonged irritation of the 'pressor' nerves, Yandell Henderson's acapnia theory, Boise's hypothesis of primary cardiac failure causing a fall in blood-pressure, and Meltzer's belief that the functions of the spinal cord were inhibited by the stimulus of injury. Since the war, Crile,[67] after extensive researches undertaken by himself and a considerable staff, both in America and at military hospitals in France, put forward the hypothesis that " the body as a whole was an electrochemical mechanism, the positive pole being the brain, the negative pole the liver, the connecting wires the nerves, the salts in solution the electrolytic fluid in which the electrochemical mechanism was immersed ". Shock was alteration of electrical conductivity by impairment of the physical structure of the cell. Dale,[68] in his Oliver-Sharpey Lectures, described the presence in shock of stagnation of blood in the capillaries, and suggested that this may be produced by the circulation of certain toxic proteins. Cannon and others believe that injury to any part of the body sets free substances into the blood-stream, which cause this dilatation of the capillaries, thereby diminishing the return of blood to the heart, with a consequent fall in blood-pressure, although the arterioles are contracted.

We see that shock is produced by extensive injury to any of the tissues of the body, and particularly, therefore, to those such as the stomach, which are richly supplied with nerves or with blood-vessels. When the ill-effects of chilling and severe loss of blood are added to those of surgical trauma, marked predisposition to any process of general infection, such as pneumonia, must result. With this view most observers agree. In a letter to my colleague, Dr. McCardie, Sir Berkeley Moynihan emphasized the importance of handling all tissues most gently if lung complications were to be avoided.

Reference has already been made to a case of Cæsarean hysterectomy under spinal analgesia for profuse concealed intra-uterine hæmorrhage. Here no general anæsthetic was employed, the operation was rapidly performed, and harmful sensory impulses were blocked by the spinal anæsthetic. By far the most disturbing influence was the tremendous loss of blood both before and during the operation. One feels compelled to attribute the subsequent pneumonia chiefly to this factor. F. B., also mentioned previously, a woman of anæmic appearance, was admitted to the gynæcological wards with the diagnosis of 'ruptured tubal gestation'. Examination of the chest revealed widespread capillary bronchitis. The abdomen was opened under spinal analgesia, and a large quantity of blood was found throughout the peritoneal cavity. Salpingotomy having been performed, the blood-clot was removed. Next day bronchopneumonia was demonstrated in all lobes, but, happily, the patient recovered. In this instance loss of blood and irritation of the peritoneum

were the potent factors which, even before operation, had laid the foundation for the subsequent pneumonia.

Too often we forget the profound chilling of a patient's body which occurs during a long operation, while more or less exposed on an unheated operating table in a cold theatre. The man who gets caught in the rain on a cold winter's day, and then develops pneumonia, has not run nearly so great a risk. Some years ago, Robb[70] made a series of observations on the fall in patients' temperatures during operation, (*a*) on a cold table, (*b*) on a table heated by means of electric lamps placed under the table.

His results were as follows: 38 abdominal sections, room at 83°, no lamps, cold table, total fall of 25·8°; 38 abdominal sections, room at 78°, with lamps, heated table, total fall of only 10·6°.

Thus, in the second series, though the room was colder, the heated table considerably reduced the cooling of the body.

Vierordt, quoted by Isabella Herb, states that 11 calories per hour are lost by the normal lungs, but 90 calories per hour are lost by the skin; so that we must endeavour to prevent loss of heat by the skin.

Corlette[71] pointed out that the loss of heat by convection from the body depends on the free movement of air over the skin. Warmth then will be conserved provided that the covering be air-tight, free from gaps. Dr. S. R. Wilson[72] has suggested an operating suit embodying this principle.

Dr. McCardie suggests that the free perspiration and the red flush produced in the skin during the administration of ether must lead to particularly rapid loss of heat, so that patients who are under the influence of ether must be kept dry and well covered.

Lastly, the custom of purging patients before operation lowers the blood-pressure, but pre-operative rectal salines are most helpful.

Chilling of the Body during Transit back to the Wards and during Recovery from the Anæsthetics.—" It is a cold wind with a moist atmosphere which chills you to the bone, and finds out the weak spots in your anatomy ". In these words Sir James Barr[73] indicates the determining factor in many a case of bronchitis or pneumonia.

After operation the patient, often imperfectly clothed, and bathed in perspiration, is taken on an ambulance along cold corridors, and maybe in a draughty lift, to the ward, where the bedclothes are tossed off during the restless recovery, and, owing to the shortage of nurses (particularly at night), they are not immediately replaced. Dr. McCardie has reminded us that the thermic centre is out of balance in these cases, and this leads to failure to conserve heat. Recently Dr. Mennell told me of an epidemic of lung complications after operations which had recently occurred at St. Thomas's Hospital. At the time of the epidemic, the hospital was undergoing alterations which necessitated the transport of patients through long corridors, where they were exposed to outside weather conditions. Dr. Mennell attributed the epidemic to the chilling of the patients at this time.

Dr. Lamb has remarked that, in order to prevent chilling of the patient during the stages of preparation, operation, and recovery, great care must be exercised in regard to detail, otherwise the unconscious patient may quite readily undergo loss of heat to a fatal extent. As mentioned above, Corlette

insists on the need of carefully covering the patient so that no draught can play on the skin. Draughts give rise to vasomotor disturbances, and in the conscious patient this leads to shivering, but under anæsthesia, although the risk is greater, no danger signal is forthcoming.

Dr. Silk in 1900 considered it dangerous to place the patient's bed in a draughty part of the ward, and I understand that he is still of this opinion. At the General Hospital, where the plenum system of ventilation is in use, Dr. McCardie suggested to me that the breeze produced by opening the door of the ward led to an abnormal number of colds and lung complications among the patients who occupied those beds over which the current of air swept. He specified the second, third, fourth, and fifth beds on the right of the ward, counting from the entrance doors. Thereupon, in several of the wards, I conned the sisters' daily reports for the year 1923. But coughs, colds, and even pneumonia, so far as I could see, did not threaten the occupants of certain beds more than the patients in others. Nevertheless, a strong draught is not desirable, and Dr. McCardie is widely supported in his advocacy of small, warm rooms, with open fires and closed windows, for the first twenty-four hours after operation.

Bearing in mind this need of perpetual vigilance by a skilled nurse, and of still air at a constant temperature, some hospital authorities (e.g., at the Bristol General Hospital) have instituted small recovery wards in immediate proximity to the operating theatres, where these conditions may be observed for twenty-four hours after operation. The results are not, as yet, conclusive, and I understand that this is mainly due to difficulties in organization.

4. THE PATIENT'S GENERAL CONDITION.

Influence of Sex.—When pneumonia occurs in patients who have not undergone surgical operations, it is found that men are more frequently attacked than women. Osler stated that he had records of a series of the lobar variety in which 533 men had suffered, but only 125 women, that is to say, a ratio of about 4 : 1. He did not, however, mention the influence of sex in the causation of bronchopneumonia.

At the Birmingham General Hospital, during two years, secondary bronchopneumonia was found in 89 autopsies from the medical wards. Of these, 57 were male and 32 were female, giving a ratio of approximately 2 : 1. Of the post-operative autopsies, bronchopneumonia was found in 104 men and 34 women, i.e., 3 : 1. (It must be pointed out that the numbers of operations performed on men and women at the hospital are about equal.) When, however, we examine the results of operations on the upper abdomen, as demonstrated by my series of 222 operations on the stomach, we find that after 165 operations on the stomach in men, bronchopneumonia occurred in 22 (13·3 per cent); and after 63 operations on the stomach in women, bronchopneumonia occurred in 2 (3·1 per cent). Supposing that equal numbers of operations had been performed on men and on women respectively, we should get pneumonia occurring in the ratio of more than four men to one woman.

In the literature, opinions on this point, as on other questions, are divided. Anders[7] said that sex was not a factor, and the chairman of the Surgical Congress of the American Medical Association in 1909 stated that "age and

sex play no part"; but Whipple has records of 128 cases of bronchopneumonia after operations on men, as compared with 36 cases of the disease in women. Isabella Herb, in 1922, published a careful analysis of 107 cases of post-operative lung complications, 76 in men and 31 in women. All the lung complications occurring after operations on the stomach and appendix were in men; after gall-bladder operations, 6 were in men and 4 in women; and after hernia operations, 8 were in men and 1 was in a woman. The remaining cases were either septic or terminal. On consideration of the relatively high frequency of bronchopneumonia after operation on men's stomachs, both in my series and in those of others, one must conclude that men are particularly susceptible to bronchopneumonia when the upper abdomen is injured.

It is reasonable to suggest that the abdominal or diaphragmatic type of respiration in men plays some part in determining the onset of bronchopneumonia. Thus, during upper abdominal operations a man sustains direct and reflex shock to the diaphragm; this leads to partial paresis of that muscle. After recovery from the anæsthetic the patient finds difficulty in coughing and in deep breathing, because the diaphragm is weak and the abdominal respiration leads to increased movement and tension in the wound. Women are not so seriously troubled, for adequate costal breathing does not disturb the abdominal wound, and even coughing may be indulged in without serious pain.

Influence of Age.—J. M. Anders, writing in 1898, considered age to be an unimportant factor, but Silk[6] pointed out that most cases of bronchopneumonia after operation occurred in persons under 40 years of age. In an analysis of his series, Whipple[17] found that in each of three years this complication occurred most frequently in patients between 30 and 40 years old, but, according to Cutler and Morton,[44] old age gives a strong predisposition.

The age incidence in my series of autopsies is shown in *Table III.*

Table III.—Age Incidence of Pneumonia in Medical and Surgical Cases.

Age in Decades	Number of Surgical Cases with Pneumonia	Order of Frequency	Number of Medical Cases with Pneumonia	Order of Frequency
0– 9	19	4	23	1
10–19	16	7	11	3
20–29	15	8	10	5
30–39	17	6	14	2
40–49	30	1	11	3
50–59	22	2	10	5
60–69	21	3	9	7
70–80 and upwards	19	4	1	8

Thus, if any inference can be made, we may notice that, in the surgical cases, pneumonia was commonest in the latter half of life; while, in the medical autopsies, the younger subjects had been more frequently attacked. But hypostatic pneumonia in the aged, and the greater severity of operations

performed on those in middle life, may account to some extent for the relatively high incidence in the more elderly. These figures do not differ greatly, and we conclude that pneumonia may complicate operations performed at any age.

5. GENERAL SYSTEMIC DISORDERS AFFECTING METABOLISM AND ORGANS OF DEFENCE.

Nutrition.—In general, surgeons find that chest complications occur more frequently after operations on hospital patients than in private practice. To some extent we may attribute this to the state of nutrition, and it is of interest to consider a series of operations at the Birmingham Children's Hospital, performed on wasted infants for congenital hypertrophic pyloric stenosis. These cases were under the care of Dr. Leonard Parsons, and the results were embodied in his Goulstonian Lectures[74] for 1924. At this hospital there is a ward for private patients, but the conditions of operation, nursing, and treatment are the same for both 'private' and 'hospital' cases. In a series of 70 infants operated upon, of whom the majority were treated by the same surgeon and the same anæsthetist (Mr. Seymour Barling and myself), 59 were from the poorer classes, and 11 were private cases. The death-rate for the poorer-class patients was 56 per cent, but there were no fatal cases among the better-class babies. Bronchopneumonia occurred with fatal results in 5 of the hospital cases, but there was no instance of this complication among the infants from the private wards. It was very evident, from the weight and state of nutrition of the babies, that the well-to-do parents had secured diagnosis and treatment at an earlier stage than the working-class parents, and to this more advanced wasting I think we may ascribe the much higher post-operative morbidity and mortality in the poorer-class infants.

Phagocytosis.—In an unpublished research on prognosis in suppurative appendicitis, Dr. Piney, the present Director of the Charing Cross Hospital Pathological Department, concluded that the presence of leucopenia at the time of operation foreshadowed delayed recovery or death, but, on the other hand, a high leucocyte count justified an optimistic prognosis. He suggested that, in patients with leucopenia, the pre-operative administration of sodium nucleinate would induce a protective leucocytosis against pneumonia. However, working on cats, certain observers have produced leucocytosis by this means, but they state that the leucocytes are not active phagocytes. Unfortunately, in routine practice the method is not practicable, and I have not had any personal experience.

Drugs.—The phagocytic action of the blood is diminished by the presence of ether, but the rectal administration of olive oil is said to re-activate the phagocytes.[46] This deleterious action of ether (and chloroform) seems to last but an hour or two.

In the case of chronic alcoholism, however, there is much evidence that the defensive mechanism of the body is seriously crippled. Capps and Coleman[28] have published an interesting analysis of 3422 cases of lobar pneumonia at the Cooke County Hospital, Chicago. These cases occurred during two periods, namely 1911–17, before prohibition was enforced, and the two years after prohibition, 1921–22. They found that the death-rate

in moderate drinkers was much higher than among abstainers, and that in excessive drinkers the death-rate was highest of all. Speaking as an anæsthetist, I should say that men who have persistently drunk alcohol to excess require large amounts of the anæsthetic agent to anæsthetize them, that they are much exhausted after operation, that they readily catch cold, and easily develop pneumonia, and that, when this happens, the death-rate is very high.

Cachexia.—With the onset of old age, the physiological processes of the body lose their vigour, the germs of pneumonia readily obtain a foothold, and then the outlook is always most grave.

Anæmia leads to abatement of the body's activities with similar results, and, on looking through the medical post-mortem records, this is confirmed.

Cachexia from Cancer.—In the majority of cases cancer is terminated by the onset of bronchopneumonia. Läwen[9] recorded pneumonia in 36 per cent of his series of gastrostomies for cancer of the œsophagus, but only in 6·6 per cent of resection of the stomach.

In my list, of 5 autopsies on patients who died after gastrostomy for cancer, bronchopneumonia was present in 4. General anæsthetics had been administered to 3, all of whom had had bronchopneumonia, but the other 2 subjects had been operated on under local analgesia, and 1 of these exhibited bronchopneumonia.

In general, it is my experience that, although local anæsthesia does perhaps reduce the rate of incidence, the majority of gastrostomy cases which are examined after death exhibit bronchopneumonia, whether the anæsthetic was general or local.

The Influence of Heart Disease and Circulatory Failure.—Elwyn[26] held that cardiac exhaustion might lead to congestion of the lungs and hypostatic pneumonia, this being one explanation of the distribution of the disease in the lower lobes. He recommended the early use of digitalis, and this is now the custom in the clinics at Vienna. Mandl[22] administers the drug within fifteen minutes of the end of the operation. Kellogg,[32] too, believes in the danger of pulmonary congestion. After operation he repeatedly applies cold compresses to the chest and massages the body to encourage a free circulation of the blood. Ranzi,[66] and also Le Dentu,[10] suggest that pulmonary stasis may lead to the formation of minute emboli, and they recommend deep breathing and free movements of the limbs, commencing as soon as possible.

It might be thought that anæsthetic drugs (and especially chloroform) would have a toxic action on the heart and induce stagnation of the blood, but in fact emboli occur very rarely on the day of operation, and nearly always after the third day.

A slow pulse-rate does not appear to lead readily to pneumonia. I have notes of two patients with heart-block, in whom the pulse-rate was usually about 40 per minute, whom I anæsthetized for prolonged operations, without any untoward sequel.

My own view is opposed to blaming weak hearts or bad circulation for the onset of the complaint under discussion. I do not remember any case of bronchopneumonia following shockless operation on patients suffering from heart disease, even though they were prolonged.

In the post-mortem records, I find that of 33 subjects from the medical

side who had died of infection or of ulcerative endocarditis, of acute or chronic myocarditis, of mitral stenosis, or of congenital morbus cordis (excluding cases with associated renal disease), pneumonia was present only in 8. This corresponds to the pneumonia incidence for the whole series of medical autopsies, i.e., 25 per cent. In many instances, non-infected infarcts were present in the lungs, but there was no pneumonia.

6. THE PRESENCE OF PULMONARY DISEASE PRIOR TO OPERATION.

During the war, when serving in Salonika, I learnt that surgical patients previously infected with the malarial parasite often sustained an attack of malaria within the forty-eight hours following operation. Similarly, in April, 1918, during the fighting round Villers-Bretonneux, many men reported sick who were found to be suffering from malaria. These fellows had been worn out by the disheartening and exhausting retreat from St. Quentin, and, when I saw them, they had been soaked for two days by drenching rain, food was scanty, and the outlook most depressing. Every one of the men had served in the East, and gave a history of previous malarial infection. Exhaustion enabled the parasite to develop once more.

It is acknowledged that pneumonia may attack the same individual on several occasions, and we believe that each attack renders the patient rather more susceptible to the complaint.

In view of these considerations, and of numerous examples of the trouble, many authorities agree that a patient who has undergone pneumonia during the year or two prior to operation is in danger of sustaining an attack of post-operative pneumonia. Graves[64] believed this to be most important, and Chapman, and Cutler and Morton, concur.

Silk, writing in November, 1920, draws attention to the importance of a recent history of severe influenza, and *Table IV* gives support to this view :—

Table IV.

INFLUENCE OF RECENT INFLUENZA ON POST-OPERATIVE PNEUMONIA.

AT THE GENERAL HOSPITAL	1906, i.e., *before* INFLUENZAL PANDEMIC	OCT. 1918—AUG. 1919, i.e., *during* INFLUENZAL PANDEMIC	1920–1922, *after* INFLUENZAL PANDEMIC
Surgical autopsies	150	150	385
Cadavers showing pneumonia	42	66	138
Incidence of pneumonia, per cent ..	28	44	35·84
Medical autopsies	124	100	350
Cadavers showing secondary pneumonia	27	32	89
Incidence of pneumonia, per cent ..	21·7	32	25·42

Thus, in death-dealing disease, both after operation and without operation, pneumonia occurred with much greater frequency during the influenzal pandemic than before it; and even during the last two or three years the incidence was higher than it was in 1906.

The following cases which have come under my notice strengthen my belief that former inflammation of the lungs, though not detected on physical examination by the anæsthetist, readily lights up when the patient is subjected to general anæsthesia and a surgical operation.

Case 2.—T. C. B., male, age 51. Bronchopneumonia, in the summer of 1921, followed by recovery. In the spring of 1922, gastro-enterostomy was performed, pnuemonia developed within forty-eight hours, and the patient died on the fifth day. At the time of operation the patient appeared to be in good general health, and the operation was not unduly severe.

In addition to those here given, I have notes of 7 patients, all of whom recovered, who gave histories of previous attacks of pneumonia, and who developed pneumonia after gastro-enterostomy (3), hysterectomy (1), and appendicectomy (3).

Case 3.—M. C., female, age 46. In 1906, anterior gastro-enterostomy was performed with a Murphy's button. Bronchopneumonia developed in both lungs, but the patient recovered.

In 1920, a plastic operation on the stomach was followed by transient lung trouble, which did not call for much attention.

In 1922, in the presence of massive adhesions, and with great difficulty, pylorectomy was performed for a large ulcer near the pylorus. The operation took two and a quarter hours, open ether being administered for the first hour, and gas and oxygen for the remainder of the time. The patient was very collapsed at the end of the operation, and transfusion with gum solution was resorted to. However, she did not rally, and death occurred on the third day. At autopsy extensive bronchopneumonia was found in both lower lobes. (There were no coughs or colds in the ward at that time.) When the patient was first seen at the hospital during an acute attack in 1906, before any operation was performed, the pulse-rate was 56 to 60 per minute. Treatment by rest in bed with a suitable diet caused the pulse-rate to rise to 76 to 80.

I regard this case as an instance of vagal reflex action from the stomach affecting the heart, and probably promoting some degree of bronchiolar spasm. However, I shall return to this point when considering bronchiolar spasm and collapse of the lungs.

The combination of heart disease (such as mitral stenosis) and a history of previous attacks of pneumonia appears to be very important.

Case 4.—S., female, age 32. History of three or four attacks of broncho-pneumonia. Double mitral disease. Primigravida in poor condition, Cæsarean section performed under open ether at term. Delivered of twins. Operation was quickly done in twenty minutes, with only slight loss of blood. Bronchopneumonia appeared at both bases on the third day, and death occurred on the seventh.

It is agreed that patients who have suffered from phthisis may undergo exacerbation of the disease as the result of operations.

Case 5.—A. B., male, age 25. History of tuberculosis, healed patch in right apex. Uneventful operation for cure of right inguinal hernia, and appendicectomy, under open ether. He commenced to cough on the third day, and tubercle was found in sputum. The cough disappeared by the tenth day.

The following notes on two patients of the same sex, type, age, who were nursed in adjoining beds, and who underwent similar operations under similar anæsthetics on the same morning, afford striking testimony

to the importance of old-standing lung disease in the determination of pneumonia after operation.

Case 6.—W. P., male, age 40. Previous history: healthy, spare build, good general condition. Duodenal ulcer. Gastro-enterostomy and appendicectomy were performed uneventfully under open ether. Collapse of part of the right lower lobe appeared on the fifth day (confirmed by X-ray examination), but no pneumonia could be found. Symptoms: slight cough, good colour. Pulse-rate never above 88, remittent temperature to 101° on third to seventh days inclusive. Respirations did not exceed 28 per minute. Quite well on the tenth day.

Case 7.—G. H., male, age 35. Bronchopneumonia eight months previously. Good general condition. Duodenal ulcer. Gastro-enterostomy and appendicectomy were performed uneventfully under open ether. 'Chesty' after operation. Definite right basal bronchopneumonia on the fourth day. Recovery.

I think that both these men suffered from collapse of the lower lobe of the right lung, but pneumonia developed only in the lung which contained the remains of old infection.

7. THE PATIENT'S RESPIRATORY SYSTEM.

The Influence of the Shape of the Chest —For the most part, the shape of the chest can have but an indirect influence in the causation of pneumonia, and any disease of which the shape of the chest is symptomatic will have the more marked effect.

In emphysema, however, where respiration is often carried out entirely by the diaphragm, interference with the free movement of this organ, as in operations on the upper abdomen, may lead to hypostatic pneumonia and, it may be, to collapse of the lungs. It should be noted that closed methods of administration of anæsthetics to emphysematous patients, in particular, will tend to defective aeration and consequent impairment of the physiological activities of the lung tissue.

The Influence of Chilling of the Lungs.—The entry of cold air into the lungs acts in two ways.

a. The body as a whole loses a considerable amount of heat. Isabella Herb,[21] quoting Vierordt, says that normal lungs lose 11 calories per hour, but the skin loses 90 calories of heat in each hour. Owing to the number of variants involved in the calculation, I think these figures give only a general indication. Corlette, in the article already quoted, points out that cold damp air chills the lungs less than warmer air which is dry, for much heat is lost in the evaporation of water to saturate the dry alveolar air with water vapour.

b. Joss[76] found that, during anæsthesia, the inspired air was at least 33° below room temperature, and he considered that the chilling reduced the bactericidal action of the lung and slowed the movements of the ciliated epithelium.

I have already mentioned the danger of inspiring a fog of droplets of ether, which must cause serious interference with the normal action of the alveolar epithelium, both by chilling and by chemical action. Shipway, in strong support of the warming of anæsthetic vapours, says that the warming of ether vapour prevents its reputed irritation of the lungs.

Silk,[75] at Malta, found that hot open air in the operating theatre and

afterwards went a long way towards abolishing lung troubles. Operations in one of the theatres which was ill-ventilated, and heated by gas fires, were frequently followed by lung complications. Silk considers therefore that the temperature and quality of the inspired air are of first importance.

The Influence of Free Secretion.—In many persons, the inhalation of ether vapour for anæsthetic purposes induces free salivation and secretion of watery mucus by the mouth, nose, and pharynx. (Some individuals, notably infants, do not secrete so vigorously.) This secretion is accompanied by a profuse outpouring of sweat by the skin, and many authorities contend that the evil effects of ether lie in aspiration into the lungs of germs contained in mucus from the nose and mouth, and in the loss of bodily heat occasioned by the evaporation of sweat. There is much to be said for this view, and the practice, now almost universal, and certainly most valuable, of injecting $\frac{1}{100}$ to $\frac{1}{75}$ gr. of atropine half an hour before operation, has prevented the secretion; yet post-operative lung troubles still occur. On the contrary, one sees ill-prepared patients in the out-patient theatre 'frothing' vigorously under ether anæsthesia, the atropine injection having been omitted; but pneumonia in these and similar cases is extremely rare.

Nevertheless, the dry patient is warmer, more readily anæsthetized, of a better colour, and far less shocked after the operation: all of which must militate against lung infections.

The Influence of Patency of the Upper Air-passages.—We all agree that a clear airway is essential to effortless respiration, but laryngeal spasm, or a congested tongue, or falling back of the jaw, may start the vicious circle which leads to cyanosis, and so back to more spasm or more congestion. Obstruction to respiration entails great fatigue of the muscles of respiration, and hinders the elimination from the blood of waste products, thus leading to cardiac exhaustion and pulmonary congestion. But, notwithstanding the care with which anæsthetists remedy this defect, pneumonia does occur after uneventful and effortless anæsthesia, while badly administered anæsthetics with cyanosis do not raise the frequency of pneumonia to any remarkable extent.

We must conclude that partial obstruction of the airway is not a primary cause of lung trouble.

The Effects of Interference with the Movements of Respiration.—The most direct means of limiting respiratory movements is by mechanical compression of the thoracic wall. Thus, after some operations, the chest is constricted by an abdominal binder. Dr. Flemming, of Bristol, told me that he had seen a patient, who had just undergone laparotomy, bound up so tightly that he was blue and nearly pulseless for an hour or more. Loosening the binder led to instant relief and a normal appearance, and lung trouble did not follow. Again, the majority of patients upon whom the modern radical method of removing a carcinomatous breast is performed awaken to find that the wound has been covered by skin stretched so tensely as to resemble parchment, and that the chest is held in a vice-like grip. Similarly, the free rise and fall of the diaphragm is obstructed by marked increase in bulk of the abdominal contents, e.g., meteorism, large ovarian cysts, pregnancy, ascites.

Now, several authors have suggested that limitation of movements by the chest wall from any cause whatsoever is a potent factor in the production

of pneumonia. With this general view of the matter one cannot entirely agree. If we consider the results of mechanical constriction of the thorax produced by the methods outlined above, we must confess that pneumonia is very rarely seen after the most extensive operations on the breast, however firmly the thorax be compressed by tight stitching; nor does pneumonia appear as a frequent complication of abdominal ascites, pregnancy, or meteorism in the absence of an operation. To my mind, then, although pulmonary hypostasis is promoted by this means, some degree of limitation of movement of the chest does not of itself lead to massive collapse of the lungs.

Massive Collapse of the Lungs.—As is well known, the credit for the pioneer work on this subject is due to William Pasteur.[77, 78, 79] In his Bradshaw Lecture of 1908, Pasteur said that cases of post-diphtheritic paralysis of the diaphragm developed airlessness, more or less complete, in one or both lower pulmonary lobes.

Keith[80] had shown that the upper lobes were distended principally by movement of the ribs, while descent of the diaphragm expanded the lower lobes. In Pasteur's opinion, paralysis of the intercostal muscles, or of the diaphragm, on one or both sides, led to deficient expansion, and often to complete airlessness of the corresponding lobes. Together with other observers, including D. J. Harries,[81] he records the frequent occurrence of partial or complete diaphragmatic paresis, more usually of the right leaf, as a sequel to laparotomy.

The diagnosis of post-operative partial diaphragmatic paresis is not easy. The most reliable means, radiography, is rarely available. In most hospitals, the portable apparatus for ward work is not of sufficient power to permit of screening. Furthermore, as a routine, one is not justified in disturbing the patient immediately after operation by the journey to the radiologists' department, in order to make an examination which may be chiefly of academic interest. Clinically, before there is any marked change in the lung, we may bear in mind that relaxation of the right leaf of the diaphragm leads to a rise in the liver dullness, and relaxation of the left half of the diaphragm permits the heart to move towards the left clavicle, while paresis of the intercostal muscles allows of descent of the liver, or of the heart, as the right side or the left side, respectively, is affected.

In this connection, the presence of Hoover's[82] sign may be sought. Normally, on inspiration, the subcostal angle is widened, because the intercostal muscles, in their efforts to broaden the chest by the bucket-handle movement of the ribs, gain the mastery over the diaphragm, which tends to narrow the thorax, but which, owing to its curved form, acts under mechanical disadvantage. Should, however, the diaphragm be paralysed, the unopposed intercostal muscles cause the chest to be even broader, and on inspiration the subcostal angle will widen to an exaggerated extent. Paralysis of the intercostal muscles, on the other hand, allows of unhampered action by the diaphragm, and inspiration narrows the subcostal angle. If paralysis is unilateral, these abnormal movements are limited to the affected side. However, in practice, one-sided paresis renders the sign somewhat uncertain. We may note that bilateral diaphragmatic paralysis gives rise on inspiration to recession of the epigastrium as contrasted with normal protrusion.

Many careful observers agree that relaxation of the diaphragm does take place with great frequency in any abdominal condition entailing pain sufficiently great to cause reflex spasm of the muscles of the anterior abdominal wall, whether the source of pain be peritoneal inflammation or an operation wound. Usually the right half of the diaphragm alone is involved. The supper eight intercostal muscles are rarely disturbed, although, for reasons I shall suggest, spasm of the ninth and tenth intercostal muscles may occur.

By what mechanism is this change in the action of the diaphragm brought about? In other words, have we to deal with direct injury to the diaphragm, or is there reflex inhibition? Relaxation of the diaphragm has been recorded in many cases of operation for appendicitis or for radical cure of an inguinal hernia, although the neighbourhood of the diaphragm had not been explored. Almost certainly, we have to deal with a reflex inhibition of which one or both of the phrenic nerves is the efferent path.

There are differences of opinion as to the afferent nerves involved. Pasteur suggested that the vagal afferent fibres were implicated; but there is no proof that stimulation of the central end of the divided vagus can produce reflex inhibition of the diaphragm; nor are extensive operations on the neck, which involve much handling of the vagus, responsible for diaphragmatic paresis, although persistent vomiting, due to stimulation of the vagus, often follows these operations. Hardy, and also Harries, arguing on Sherrington's law of synergic co-operation between a muscle and its antagonist, very reasonably suggest that increased tone of the protective muscles of the anterior abdominal wall is excited by inflammation such as appendicitis and peritonitis, or by a surgical wound, and that the diaphragm, which in this instance is the antagonist, is relaxed. That this does in fact occur is the experience of surgeons when they open the abdomen for perforation of a gastric ulcer. One finds that the stomach and bowels lie limply on the posterior wall, almost without the usual movement due to respiration, and it is rare indeed, before meteorism has set in, that the surgeon has trouble with protrusion of the abdominal contents through the wound. This phenomenon, I think, must be due to abnormal quiescence (i.e., inhibition) of the diaphragm. According to this hypothesis, the afferent paths are the sympathetic sensory fibres from the inflamed area.

Before leaving the subject, I will recall two cases which presented points of interest.

Case 8.—**T. M., Male, age 50. Perforated duodenal ulcer.**

Grave condition, complicated from the anæsthetist's point of view by enormous bilateral adenomata of the thyroid. High injection of spinal anæsthetic was administered into the ninth dorsal space. The operation proceeded uneventfully until the surgeon explored the under surface of the diaphragm, whereupon the patient cried, "You are hurting my right shoulder". This was an instance of direct injury to the diaphragm and of simple phrenic reflex. The patient died sixty hours after operation, and autopsy revealed œdema and congestion of the lower lobes. In this case some paresis of the diaphragm was present at operation.

Case 9.—**M. W., Female, age 55. Operation for incarceration of the bowel and omentum in a left inguinal hernia sac.**

The presence of severe bronchitis caused me to induce spinal analgesia as high as the umbilicus. The patient talked cheerily until, as the surgeon delivered the

hernial sac, with its contents, she exclaimed, "You are crushing my chest". The discomfort was limited to the lower region of the thorax. Reduction of the incarcerated omentum and small bowel soon relieved her, but vigorous manipulation of the sac did not cause sensation of any kind.

I think the afferent path of this reflex was the sensory sympathetic fibres of the lower intercostal nerves.

H. E. Griffiths[83] recalls that the peritoneum covering the gall-bladder and the pyloric end of the stomach receives its nerve-supply from the fifth to the tenth dorsal nerves, from the right phrenic nerve, and from both vagi. Here at least are plenty of different afferent paths for reflexes to the lower intercostal muscles and to the diaphragm.

When working in France with Professor Gask, my colleague, Dr. K. D. Wilkinson, observed unilateral and even complete diaphragmatic paresis in men severely wounded by bullets or shell fragments in any part of the body. In these conditions any sensory nerve may supply the afferent path of the reflex under discussion.

Having established the fact that relaxation of the diaphragm does most frequently occur after operations on the abdomen, we must inquire what happens to the lung whose expansion, Keith says, is controlled by this muscle.

It has been observed by Pasteur and others that often the lower lobe on the affected side becomes airless, and that the collapsed region of the lung usually produces the following symptoms and signs:—

a. Onset, sudden or gradual, occurs usually with distress on the first, second, third, fourth, or fifth day after operation.

b. Sputum of varying type appears some hours after the onset of massive collapse.

c. Mobility of the affected region is decreased.

d. There is retraction of the rib spaces over the collapsed area of the lung.

e. The breath sounds are inaudible over the diseased area.

f. Tubular and bronchial breath sounds are heard above the affected area.

g. The heart is displaced towards the side of collapse. Pasteur believes this to be pathognomonic of the condition.

h. Temperature, pulse-rate, and rate of respiration are raised.

The rate of onset may be quite gradual or it may be very sudden. And this applies to recovery. The most striking example that I have seen took a week to develop and several months to recover.

Case 10.—**A. L., Female, age 16. Right hemithyroidectomy for parenchymatous goitre.**

Uneventful operation under open ether. There was slight 'chestiness' from the first day, which gradually increased until on the sixth day the patient complained of great pain on the right side of the chest, and dyspnœa with cyanosis. Examination of the chest at that date showed: (*a*) Almost complete absence of movement on the right side; (*b*) Retraction of the right rib spaces on inspiration; (*c*) Apex beat two inches internal to the left nipple line; (*d*) Dullness on percussion up to the second right rib; (*e*) Complete absence of breath sounds over the lower two-thirds of the right portion of the chest, with bronchial breath sounds over the upper third.

Radiography showed complete paralysis of the right half of the diaphragm, with opacity of the right lower and middle lobes and the inferior half of the upper

lobe. There was ascent of the paralysed diaphragm on inspiration, and descent on expiration. Remittent high temperature and quick respiration and pulse-beat continued for three weeks, and returned gradually to normal. There was no definite bronchopneumonia. We suspected that the paresis of the diaphragm was due to some permanent lesion to the right phrenic nerve, possibly from direct injury or from extravasation of blood. I have related this case at some length, for the clinical picture was most impressive. (Later: The patient recovered. A second operation was undertaken six months later for further removal of the thyroid, under ether. Uneventful recovery followed.)

We have agreed that transient paresis of the diaphragm often escapes notice, and the same result must apply to partial collapse of the lungs, which does not give rise to subjective symptoms.

At the Middlesex Hospital, during the period 1906–12, Pasteur and Hardy found 28 instances of massive collapse of the lung, which were noted in a series of 5594 operations on the abdomen or kidneys. The majority of these cases followed sub-umbilical operations. I have records of 12 patients who developed the condition (*Table V*).

Table V.—Massive Collapse of the Lung following Operation.

Operations	Total No.	Male	Female	Side of Collapse			Day of Onset	Result
				L.	R.	Both		
Right inguinal hernia	4	4	–	–	4	–	1st, 4th 3rd, 3rd	All cured
Hemithyroidectomy	2	–	2	–	2	–	1st, 7th	Both cured
Appendicectomy	2	1	1	1	1	–	2nd, 2nd	Both cured
Gastric operations	4	3	1	–	2	2	1st, 2nd 2nd, 7th	Two pneumonia, died; one pneumonia, one collapse, lived

As is customary, only those patients died who had developed pneumonia, for massive collapse of itself is not of grave import. With regard to the site of operation, 7, including the thyroid cases, were above the umbilicus, and 5 were sub-umbilical.

In this list, I include only cases which at some stage showed lung collapse apart from pneumonia. It is extremely probable that massive collapse is the precursor of many cases of post-operative pneumonia, but the condition is often overlooked until pneumonia supervenes.

Opinions are still divided as to the cause of massive collapse of the lung. Pasteur, as we have seen, traces the course of events: abdominal section, diaphragmatic inhibition (of shorter or longer duration), collapse of one or both lower lobes of the lungs, and then, in some cases, pneumonia. Elliott and Dingley,[84] however, on clinical and experimental grounds, believe the starting-point is plugging of the lesser bronchioles with mucus, and a generalized bronchitis. Then the air in the associated alveoli is absorbed (Lichtheim's experiments[85]) and the lung collapses, drawing the heart towards the shrunken lung. These observers conceive that arrest of unilateral or bilateral diaphragmatic or intercostal movement may occur from reflexes of intrathoracic origin.

Important as these suggestions are, one is induced to adhere rather to Pasteur's view. On Elliott and Dingley's hypothesis, we should expect bronchitis to be common, and pneumonia to occur at any region of the chest. But capillary bronchitis is far from common after operations, whilst pneumonia in these cases is rarely diffuse, and nearly always in the lower portion of the lung.

A further suggestion has been made by various observers, who consider that spasm of the bronchial musculature may lead to secondary collapse of the lungs, as shown experimentally by Brodie and Dixon.[86] Colour is lent to this view, if we reflect on the vagal reflex by which, in an analogous manner, gastric inflammation lowers the pulse-rate, and successful treatment of a gastric ulcer raises the pulse-rate. When looking through some volumes of records of medical cases, I found that, in the absence of true heart disease, every pulse-rate of less than 60 per minute was associated with gastric trouble. If, however, bronchial spasm were an important etiological factor in the production of massive collapse, asthma should be a common forerunner. But asthma is not a precursor of massive collapse or of pneumonia. On the contrary, it is my experience, and Dr. Haldane and Professor Jonathan Meakins[58] agree, that asthmatic patients take anæsthetics extremely well, and usually are spared renewal of attacks for at least a week after a general anæsthetic.

I have been asked how massive collapse can predispose to pneumonia, if it is only 'airlessness of the lungs'. In reply, I deny that, in my view, massive collapse is a purely mechanical phenomenon; for there is always pyrexia and increase in pulse-beat and respiratory rate, and usually muco-purulent sputum. To my mind, the most satisfactory explanation which will fit in with observed facts and established theories is as follows:—

Pain in the abdomen from operative trauma, or from inflammation, leads to rigidity of the abdominal wall and to reflex inhibition of the diaphragm, and perhaps to some degree of spasm of the lower intercostal muscles. The lower lobes of the lungs then do not freely expand and contract, so that congestion with some œdema sets in. The bronchioles of the disordered region are more or less choked by this swelling of their walls, which has been increased from irritation by the anæsthetic agent. Into this motionless and well-nourished region, germs and particles of débris arrive, either by the air or by the blood or lymph. In these cases the patient cannot cough freely, and the débris, which should be expectorated or at least evacuated by the ciliated epithelium, lie undisturbed. Closure of the bronchioles and absorption of air from the alveoli now ensues (Elliott and Dingley).

The result of this process is massive collapse of the lung, or, according to Whipple, pneumonitis. When the infection is particularly virulent, the patient's resistance is low, and the diaphragmatic paresis persists. We shall not be surprised to find true bronchopneumonia as the sequel, and it will be of coalescing lobular distribution following the bronchiolar blocking which was the determining factor of the lung collapse.

8. THE PROCESS BY WHICH THE LUNG BECOMES INFECTED.

Effect of Season.—In most years, experience shows that patients run greater risk of lung affection if they undergo operation in the winter months. Läwen found that 70 per cent of his series of post-operative lung infection

occurred in the winter, and Armstrong's results agree with this. Stuart Ross,[36] working in Macedonia, recorded 21 cases of post-operative pneumonia; 11 of these occurred between the middle of December and the end of February. Whipple, however, in each of three consecutive years, namely, 1915, 1916, and 1917, observed that March was the month with the heaviest incidence. In 1921, 1922, and 1923, my clinical records and the post-mortem findings show that the end of November and beginning of December, together with the months of February, March, and April, were the worst periods.

At Vienna, in the winter 1919–1920, during the great shortage of coal and food, Mandl's clinic received a heavy visitation of lung trouble, which appeared in 27 per cent of the operation cases. Mandl used digipurat in large doses with satisfactory results, for the incidence dropped to 8 per cent.

The Influence of Country and Town.—At the Anæsthetic Section of the British Medical Association Meeting at Glasgow in 1922, Mr. H. J. Patterson affirmed his belief that operations of choice should be performed in the country. The hospitals in the city should be reserved for urgent cases. In support of this contention Mr. Patterson supplied records of two series of cases, one of 150 operated on in the city, and the other of 150 country operations. The surgeon, anæsthetist, and anæsthetic agent being the same for all the patients, he showed that the average post-operative rise of temperature was one degree higher in the town series than in the country list.

Nevertheless, while agreeing that country air must be less heavily laden with germs, one not infrequently hears of post-operative pneumonia attacking patients in country cottage hospitals.

In the course of this essay, mention has been made of the work in operating centres all over the world. Not one, with knowledge of the facts, can claim to be free from this scourge.

The Effect of Epidemics.—If there be proof of the increased prevalence of post-operative lung complications during epidemics of coughs, colds, and influenza, surgeons will do well at those times to postpone less urgent operations until better conditions prevail. When doctors in general practice are busy with widespread catarrhal troubles, influenza, and pneumonia, I have sometimes noticed simultaneously an outbreak of post-operative pneumonia at the hospital. In February, 1924, colds were raging everywhere, and one morning 3 fresh cases of pneumonia were reported from the surgical wards. In November and December, 1921, during the course of a fortnight, 6 cases of pneumonia occurred after operations, and I was informed later by Dr. Ashley Daly that during the same period at the London Hospital, chest complications had become so frequent that some of the surgeons postponed operations wherever possible.

Gynæcological operations rarely lead to pneumonia (2 cases in 110 patients), but during the season of epidemic catarrh the gynæcological ward at the General Hospital with 12 beds claimed 4 victims to acute bronchitis after operation. All recovered, but, had the operation involved the epigastrium, dangerous pneumonia might have been the sequel.

I expended some considerable time and energy in the preparation of a table to show the incidence of pneumonia in medical and surgical autopsies for each month in the two years, May, 1920, to April, 1922. The numbers

were too small for inclusion here, but in general terms February, March, April (when catarrhal conditions are rife) were the months of highest incidence of secondary pneumonia in the medical and surgical cadavers, and it is interesting to find that in these months there were most autopsies on primary pneumonia.

In my series of 222 operations on the stomach, 24 were complicated by pneumonia. Of these, 11 occurred in February, March, and April.

Attention has already been drawn to the relatively frequent appearance of secondary pneumonia in the surgical wards during the influenzal epidemic of 1918.

The Quality of the Air in the Ward.—At the General Hospital, ventilation is effected by the plenum system. This method produces a still and enervating atmosphere, which is particularly oppressive in the smaller rooms. Many of the staff attribute post-operative pneumonia to this cause, and point out patients who, though seriously ill in a side ward, have quickly recovered when placed on a balcony in the fresh air.

But the post-operative incidence is not more serious at the General Hospital than it is at the Queen's Hospital, where fires and open windows are employed, and I refer the reader to the comparison of the post-mortem findings given in *Table II*. Yet, physicians rightly agree that well-warmed fresh air is essential for the nursing of pneumonia.

The next question is: Is the air from the long and dusty channels of the plenum system laden with pathogenic organisms, such as the pneumococcus or tubercle bacilli? Dr. Hillier and I set out to collect evidence on this point. Asbestos wool was packed lightly into a wide silica tube. This was sterilized in a Bunsen flame and, without touching the wool, the apparatus was placed in a hole in a stiff sheet of cardboard, and the cardboard was fixed in a ward, over the opening of one of the plenum air-inlets. After an interval of from three days to a week, the asbestos wool 'catch' was immersed in broth. Cultures from the emulsion thus formed did not grow any organism of pathological importance. Some of the broth was injected into guinea-pigs and into mice. We performed the experiment three times. The guinea-pigs were not affected, but on two occasions a mouse died. Dr. Hillier found a coliform bacillus in the heart's blood of each of these animals, but no pneumococci, streptococci, or tubercle bacilli were found.

As these investigations were undertaken when coughs, colds, and pneumonia were present in the ward, we decided that the plenum air was safe.

Infection by Aspiration of Material from the Upper Air-passages and the Mouth.—When my attention was first drawn to the subject of this essay by Professor J. Shaw Dunn, then Professor of Pathology in Birmingham University, he said that, in his opinion, aspiration pneumonia was the commonest form of post-operative pneumonia, and was identified at post-mortem by the presence of multiple suppurative foci in the lung. Läwen records 180 cases of pneumonia in a series of 9755 operations. Of these cases, 40 were due to aspiration of material into the lung. Certainly, until the introduction of intratracheal methods, aspiration pneumonia was chiefly responsible for the high death-rate after operations for excision of cancerous growths in the mouth, pharynx, nose, or larynx.

Many authors agree with these views. Von Grossman[87] suggested that,

during an attack of rhinitis, ether vapour 'skinned off' infected material from the nose, and this was aspirated into the lung. Darnall[62] thought microbes from infected air-sinuses might lead to the trouble. Cutler and Morton blamed inhalation of nasal mucus and pus from septic teeth and gums. Wight considered that atropine, by drying up the nasal and oral secretions, reduced the danger to the patient.

The hypothesis is employed to account for many of the points observed. Beckman[13] thus explained the almost invariable lobular distribution, and Schultz,[8] on anatomical grounds, said aspiration would lead more readily to infection of the right lung (the usual sufferer). By experiments on animals, Cooper[89] showed that dry soot placed in the larynx reached the bronchioles, but not the alveoli. On the other hand, coloured fluids (indian ink) inhaled in the horizontal position entered the alveoli of the upper lobes, but if the animal were placed vertically the lower lobes were filled with the liquid.

During anæsthesia, when inserting the intratracheal catheter by direct laryngoscopy, I have seen mucus drawn into the larynx. If the mucus be heavily infected with pathogenic organisms, one would certainly expect lung trouble to follow. Nevertheless, recently, I saw foul-smelling pus from a large peri-œsophageal abscess enter the trachea. Although this was followed by intratracheal anæsthesia, lasting one hour, the patient did not develop so much as a cough. The abscess had been exuding pus into the œsophagus during several weeks prior to the operation, and the patient's immunity must have been very high.

The ciliated epithelium of the trachea and bronchi normally evacuate foreign particles, but profound anæsthesia of long duration, involving chilling of the mucous surface, together with the action of strong ether or chloroform vapour, must paralyse the cilia. Dr. Flemming has shown that chloroform or ether arrests the cilia of *Paramœcium* provided the concentration be sufficiently great. The action is more easily reversible with ether than with chloroform.

Coughing is Nature's safeguard against foreign bodies in the lung, and, as we have seen, operations, particularly on the upper abdomen, abrogate this function.

To minimize these dangers the author[90] has designed a special anæsthetic apparatus. It consists of a large oral airway, a funnel-shaped mask, and a gauze carrier. The airway ensures that all air inhaled by the patient shall pass through it. By boiling the apparatus before use, by inserting the airway as soon as the pharyngeal cough reflex is abolished, and by leaving it in the mouth until coughing returns, there is but little danger of aspiration. I have seen pneumonia after anæsthesia induced with this apparatus, but I am confident that aspiration troubles have been less frequent.

Air-borne Infection.—Allusion has been made to small epidemics of coughs, colds, bronchitis, and even pneumonia, which occur from time to time in the surgical wards, and these occurrences seem to prove that neighbouring cases, before or just after operation, may be the source of infection.

Whipple's researches teach us that in many patients, before operation, the pharynx contains pneumococcus *Group IV*, and if pneumonia develops, the sputum gives a culture of the same organism. Thus, without the aspiration

of mucus, patients' lungs may be infected by the organisms present in their own throats, if the protective power of the lung is lost.

Infection from the Lymph.—Läwen said that 8·5 per cent of his patients who suffered from peritonitis developed pneumonia. This high incidence he attributed to the flow of infected lymph from the peritoneal cavity through the right half of the diaphragm to the pleura and lung. This suggestion may explain the relative frequency of right-sided pneumonia in these cases, there being no direct passage for the lymph through the left half of the diaphragm. Goebel goes further, and suggests that, when opening the stomach or bowel during gastro-enterostomy, the chyle is infected by the intestinal contents, and direct flow through to the lungs is effected. But the interior of the intestine hereabouts is nearly sterile, and it is not easy to see how the chyle gets directly to the lungs.

In my opinion, lymph infection from the abdomen gives rise to pleurisy rather than to pneumonia, and the post-mortem records support this view. Empyema occasionally follows operations on an infected gall-bladder, and pneumonia in these cases is usually absent. A colleague suffering from a right-sided metastatic subphrenic abscess developed serofibrinous pleurisy without pneumonia. In the course of six months three patients, of whom one died, each showed right-sided pleurisy without pneumonia complicating convalescence from gastro-enterostomy.

Infection from the Blood-stream.—The blood may promote infection of the lung by one of three methods.

a. In septicæmia, blood laden with organisms deposits its load in the lungs. Rogers[88] investigated the bactericidal power of the normal lungs of dogs and rabbits. By injecting *B. anthracis*, or *Staphylococcus aureus*, or *Streptococcus erysipelatus* into the external jugular vein, and into the portal vein, he found that the lung destroyed these germs, but not so speedily as the liver. It is reasonable to deduce that the lung of an enfeebled, semi-narcotized patient has lost some of this bactericidal power.

b. Septic embolism. These emboli may be minute, but it is a common event in the post-mortem room to find in the cadavers of patients who have suffered from septic mastoiditis, suppurative peritonitis, or widespread cellulitis, numerous small abscesses scattered through the lungs, sometimes, but not always, surrounded by areas of consolidation. True infarction may occur.

Cutler and Morton stated that 60 per cent of their cases of pneumonia showed a septic focus, and they, together with Hunt, believe septic embolism to be the primary causal factor for which we are seeking.

For my part, on examination of post-mortem records, I have been surprised by the frequent occurrence of *abscesses* in the lung in fatal mastoid disease—probably through the spacious lateral sinus and the jugular vein. In two years, in 1000 post-mortem examinations there were 31 examples of septic emboli in the lungs.

c. Non-infected pulmonary embolism. There can be no doubt that many conditions diagnosed as collapse of the lungs, or as pneumonia, are examples of pulmonary embolism. The sudden attack of severe pain, the motionless ribs over the affected area, and the stony dullness, sometimes misguide the observer, who may diagnose massive collapse of the lung.

It is by no means a rare catastrophe. At the General Hospital fatal aseptic pulmonary embolism occurred 9 times in a series of 8000 operations of all kinds (0·11 per cent). This does not include those who died after leaving hospital. In Korte's[91] clinic at Berlin, 22 cases of pulmonary embolism were recorded in 9727 operations (0·2 per cent). Ranzi's figures are 57 in 6871 operations (0·83 per cent).

Within one week three patients whom I had anæsthetized died of the condition. The operations were for the removal of gall-stones, of a carcinoma of the breast, and of the prostate, respectively. The accidents occurred at least two weeks after operation. In the great majority of cases the accident follows operations on the pelvic organs, namely, uterus, prostate, and even hæmorrhoids (as in an old lady recently).

Lockhart-Mummery[92] and Sir Thomas Lewis blame slow circulation due to : (*a*) A weak heart, as in the aged ; (*b*) Operations among the large veins of the pelvis ; and (*c*) Loss of fluids. Lapthorne-Smith[93] states that there is excess of fibrinogen in the blood, and he recommends that great care should be taken to replace loss of fluid from perspiring, vomiting, catharsis, diuresis, and hæmorrhage. Free movements should be started as soon as possible, and digitalis will help to promote a strong heart-beat.

Should the patient rally from the primary attack, abscess of the lung, or pneumonia, which Beckman considers to be lobar in type, may follow.

CONCLUSIONS.

As briefly as possible, I now append the conclusions to which this lengthy discussion has led me :—

1. Post-operative pneumonia occurs with grave frequency.
2. Often it is not recognized, and figures which purport to give its incidence are unreliable.
3. The anæsthetic agent and the method of administration, save in special cases, seldom are decisive factors.
4. Age and sex are not of importance.
5. General health and local disease of the lungs may play a considerable part.
6. At operation, every care should be taken to prevent loss of heat, of fluid, and of blood, and especially exhaustion from trauma to nerve tissue, and to highly vascular parts.
7. Infection of the lung is often by means of aspiration in the presence of certain other factors.
8. Severe sepsis in other regions affects the lung via the blood-stream.
9. There is evidence that lymphatic infection through the right half of the diaphragm leads first to pleurisy and then to pneumonia.
10. In the absence of severe sepsis, operations on the abdomen, and especially the upper abdomen, provide the start of the chain of events which leads to pneumonia.
11. Pain in the abdomen from operative trauma, or from inflammation, gives rise to rigidity of the anterior abdominal wall and to reflex inhibition of the diaphragm, together with some spasm of the lower intercostal muscles.

The lower lobes of the lungs, then, do not freely expand and contract, so that congestion of blood with œdema sets in. The bronchioles of the disordered region are more or less choked by this swelling of their walls, which has, perhaps, been increased from irritation by the anæsthetic agent. Into this motionless and well-nourished region, germs and particles of débris arrive. The mode of access may be by the air or by the blood or by the lymph. In these circumstances the patient cannot cough owing to pain, and the exudate and the débris, which should have been expectorated or evacuated by the ciliated epithelium, lie undisturbed in the lower lobes. Closure of the bronchioles in this nearly motionless area, and absorption of air from the alveoli, now ensue (Elliott and Dingley). The result of the process is massive collapse of the lung or, according to Whipple, pneumonitis.

When the infection is particularly virulent, if the patient's resistance is low, and if the diaphragmatic paralysis persists, we shall not be surprised to find true bronchopneumonia as the sequel, and it will be of coalescing lobular distribution, chiefly in the lower lobes, affecting those lobules whose obstruction determined the collapse of that portion of the lung.

REFERENCES.

[1] Silk, *Brit. Med. Jour.*, 1897, April.
[2] Barling, Stanley, *Ibid.*, 1898, Oct.
[3] Teale, Pridgin, *Ibid.*
[4] Rumboll, *Ibid.*, 1900.
[5] Aldrich-Blake, *Ibid.*, 1900, Aug.
[6] Society of Anæsthetists, *Ibid.*, 1900, March.
[7] Anders, J. M., *Univ. Med. Mag.*, 1898, Aug.
[8] Schultz, *Med. and Surg. Rep. Presbyterian Hosp.*, 1898.
[9] Lawen, *Beitr. z. klin. Chir.*, 1906.
[10] Le Dentu, *Arch. gén. de Méd.*, 1906, March.
[11] Armstrong, *Brit. Med. Jour.*, 1906, May.
[12] Wight, *Johns Hop. Hosp. Bull.*, 1908, March.
[13] Beckman, *Mayo Clin. Rep.*, 1913.
[14] McKesson, *Jour. Amer. Med. Assoc.*, 1913.
[15] Cutler and Hunt, *Surg. Gynecol. and Obst.*, 1920, July.
[16] Vedin, *Jour. Amer. Med. Assoc.*, 1917, Jan.
[17] Whipple, *Surg. Gynecol. and Obst.*, 1918, Jan.
[18] Henle, *Arch. f. klin. Chir.*, 1901.
[19] McKesson, *Amer. Jour. Surg.*, 1918, Jan.
[20] Cleveland, *Surg. Gynecol. and Obst.*, 1919, March.
[21] Herb, *Jour. Amer. Med. Assoc.*, 1922, July.
[22] Mandl, *Deut. Zeits. f. Chir.*, 1921, clxv, 67.
[23] Homans, *Johns Hop. Hosp. Bull.*, 1909, April.
[24] Muller, *Narcologie.*
[25] Smith, G. F. R., *Brit. Med. Jour.*, 1922, April.
[26] Elwyn, *Jour. Amer. Med. Assoc.*, lxxix, No. 26.
[27] Jordan, W. R., Meeting of Sec. Anæsth., Roy. Soc. Med., 1923, Dec.
[28] Capps and Coleman, *Jour. Amer. Med. Assoc.*, 1923, March 17.
[29] Pasteur, *Brit. Jour. Surg.*, 1914, i, 587.
[30] Mason, J., *Diseases of Children*, 1916.
[31] Moore, Sir John, *Brit. Med. Jour.*, 1901.
[32] Kellogg, *Jour. Amer. Med. Assoc.*, 1920, Oct.
[33] Neufeld and Handel, *Alb. a. d. Kais. Gesund.*, 1910, xxxiv (quoted by Whipple, *see above*).
[34] Wollstein and Meltzer, *Jour. of Exper. Med.*, 1913, xvii.
[35] Hölscher, *Arch. f. klin. Chir.*, 1897, xvii, Pt. 1.
[36] Lamb et al., *Brit. Med. Jour.*, 1922, Nov. 11.

[37] Poppert, *Deut. Zeits. f. Chir.*, lxvii.
[38] Chapman, *Ann. of Surg.*, 1904.
[39] Drummond, *Brit. Med. Jour.*, 1898.
[40] Gerulanos, *Zentralb. f. Chir.*, 1901, Jan.
[41] Dupley and Hallion, *Arch. gén. de Méd.*, 1902.
[42] Crouch and Corner, *Lancet*, 1902, May.
[43] Reamy, *Boston Med. and Surg. Jour.*, 1900.
[44] Cutler and Morton, *Surg. Gynecol. and Obst.*, 1917, Dec.
[45] Marshall, "Administration of Anæsthetics at the Front", *Brit. Med. Jour.*, 1917 June.
[46] Graham, *Jour. Amer. Med. Assoc.*, 1911, Feb.
[47] Flemming, *Brit. Med. Jour.*, 1922, ii, 22.
[48] Von Lichtenburg, *Lancet*, 1906, Dec.
[49] Campiche, *Brit. Med. Jour.*, 1905.
[50] Riley, *Jour. Amer. Med. Assoc.*, 1918, Jan.
[51] Henle, *Semaine méd.*, 1901.
[52] Goebel, *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xviii, No. 1.
[53] Hendry, *New Zealand Med. Jour.*
[54] Mickulicz, *Handbook of Practical Surgery*, 1900.
[55] Griffin, *Lancet*, 1920, May 1.
[56] Rood, *Ibid.*, 1919, March.
[57] Featherstone, *Proc. Roy. Soc. Med.*, 1924, March.
[58] Meakins, Jonathan, B.M.A. Lecture, Birmingham, 1924, Feb.
[59] Hardy, T. L., Thesis for Cambridge M.B., 1914.
[60] Henderson, Yandell, *Jour. Amer. Med. Assoc.*, 1915, July.
[61] McCardie, *Brit. Jour. Anæsthesia*, i, No. 1.
[62] Darnall, *Trans. Assoc. Obst. and Gyn.*, 1916, Sept.
[63] Campbell, *Montreal Med. Jour.*, 1899.
[64] Graves, *Boston Med. and Surg. Jour.*, 1910, Sept.
[65] Van Leeuwen, *Proc. Roy. Soc. Med.*, 1924, June.
[66] Ranzi, *Arch. f. klin. Chir.*, lxxvi.
[67] International Congress of Surgery, *Brit. Med. Jour.*, 1923, ii, 190.
[68] Dale, H. H., *Ibid.*, July.
[69] Brown, H. H., *Ibid.*, 1924, i, 746.
[70] Robb, *Progrès méd.*, 1912, ii.
[71] Corlette, *Med. Jour. of Australia*, 1921, Aug.
[72] Wilson, S. R., *Brit. Jour. Anæsthesia*, 1924, April.
[73] Barr, Sir James, *Lancet*, 1924, i, 90.
[74] Parsons, Leonard G., *Ibid.*, 946 (Goulstonian Lectures).
[75] Silk, *Jour. Amer. Med. Assoc.*, 1920, Nov.
[76] Joss, *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1911, xxii.
[77] Pasteur, *Lancet*, 1908, ii, 1351.
[78] Pasteur, *Ibid.*, 1910, ii, 1080.
[79] Pasteur, *Brit. Jour. Surg.*, 1914, i, 587.
[80] Keith, *Brit. Med. Jour.*, 1908, Aug.
[81] Harries, D. J., *Practitioner*, 1923, April, 188.
[82] Hoover, C. F., *Amer. Med. Jour.*, 1920, 633.
[83] Griffiths, H. E., *Lancet*, 1923, i, 265.
[84] Elliott and Dingley, *Ibid.*, 1914, May 9.
[85] Lichtheim, *Arch. f. exper. Pathol. u. Pharmakol.*, 1879.
[86] Brodie and Dixon, *Jour. of Physiol.*, 1903, xxix, 97.
[87] Von Grossmann, *Deut. med. Woch.*, 1895.
[88] Rogers, *Function of the Lung*, 1912, Paris.
[89] Cooper, *Jour. Amer. Med. Assoc.*, 1922, June 17.
[90] Featherstone, *Lancet*, 1922, Oct. 21.
[91] Korte, *Deut. med. Woch.*, 1909, July.
[92] Lockhart-Mummery, *Proc. Roy. Soc. Med.*, 1922, March.
[93] Lapthorne-Smith, *Brit. Med. Jour.*, 1918, June.

SPONDYLITIS DEFORMANS.

By R. LAWFORD KNAGGS, London.

This is the name given to a complex group of cases which possess in common certain characteristic clinical features—namely, a painful stiffness of the spine ending in its complete rigidity, combined, in nearly all instances, with a kyphotic curvature producing a variable degree of deformity. Museum specimens obtained from such cases are usually macerated, and often, owing probably to the very chronic course of the malady, without clinical notes. Consequently their classification presents difficulties; yet by the study of the pathological anatomy it is possible to resolve these heterogeneous cases into at least three separate pathological conditions, each of which has its own peculiar clinical characters. These pathological and clinical peculiarities point to the recognition of three different affections:—

I. *Ossification of the spinal ligaments, more or less general,* is the characteristic lesion in one set. This condition may be designated *spondylitis ossificans ligamentosa.*

II. *An upper dorsal curvature and atrophy of the vertebral discs* distinguish another set. As this condition depends in the first instance upon impaired muscular power, it may be called *spondylitis muscularis.*

III. In a third set the *changes are of an osteo-arthritic* nature—*spondylitis osteo-arthritica.*

I. OSSIFICATION OF THE SPINAL LIGAMENTS.

(*Spondylitis Ossificans Ligamentosa.*)

Ossification of ligaments is the conspicuous abnormality in a very large proportion of museum specimens of spondylitis deformans. This preponderance is probably accidental, and may depend upon the striking character of the lesion.

Every gradation may be met with, from ossification of the anterior common ligament alone, to a similar change in all the spinal ligaments. Often when the spine is affected, other joints—such as those of the limbs, the shoulder girdle, and the jaw—are attacked. But the disease in the spine is peculiar to it; ankylosis there is due to this ligamentous ossification. In the other joints the ligaments do not ossify, but opposing articular surfaces become united by fibrous tissue, and eventually by bone. There are, however, certain joints in the spine which ankylose in the same way—namely, the occipito-atloid and the atlo-axoid—but in these movement is free, and there is no intervening fibrocartilage.

The condition which occurs in the joints of the extremities in association with spondylitis ossificans ligamentosa is that which, at the present time, is regarded as rheumatoid arthritis, and there cannot be much doubt that the affection which is being considered here is the form that disease assumes

when it attacks the vertebral column. The pathogenesis is also similar, the manifestations in both cases being almost certainly the result of circulating toxins. Pathologists of a bygone generation were on the right tack when they began to associate these painful rigid spines with the after-effects of gonorrhœa, but in these days it seems astonishing that they should have suspected a problematical gonorrhœa when they were in the presence of a suppurating condition of a different kind.

OSSIFICATION OF THE ANTERIOR COMMON LIGAMENT.

This may occasionally occur by itself in the human subject (St. Bart.'s Hosp. Museum 1080A), but it is usually associated with ossification of other spinal ligaments.

The ossified ligament may form a strip of bone attached along the front of a number of consecutive vertebræ, as in the spine of a celebrated Parisian character called Seraphin, which is illustrated in Hutchinson's *Archives* (vii, 246, Pl. cxxxiii; also in the Catalogue and Atlas of the Musée Dupuytren, 652A, where it is described at greater length), or may even extend the whole length of the column. But the ossific layer shows a tendency to spread laterally over one or both sides of the bodies, and in advanced cases round to the back. The posterior common ligament may also be involved. This was so in Seraphin's spine, and also in that of an old woman who had been bedridden for twenty-five years and died of intestinal obstruction. In her case every joint in her body was affected, including the temporo-mandibular and sterno-clavicular (St. Bart.'s Hosp. Museum, No. 1085c).

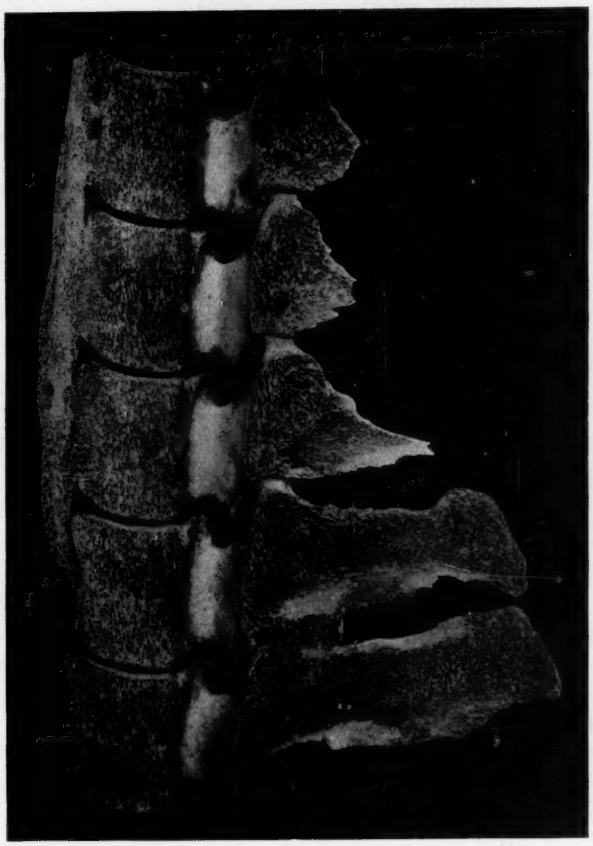


FIG. 306.—Dorsal vertebræ of a horse showing ossification of the anterior common ligament (*No.* 2054, *R.C.S. Museum*).

The tendency for the ossified layer to cover both the front and sides of the bodies may be explained by the variations met with in the anterior common ligament. It is by no means invariably a well-defined ligamentous strip over the most prominent part of the curve of the bodies, but it may cover more or

less completely their whole anterior surface. Less dense at the sides than in front, it is apt to show small elongated gaps, the ligamentous tissue being insufficient to make a complete covering (*see* description in Morris's *Anatomy of the Joints*, 72, and Quain, iv, Pt. 1, 223). A definite bulging over the position of the discs occurs in the healthy spine, but in these cases it is apt to be exaggerated, not as a consequence of osteophytic lipping for which it is very commonly mistaken, but because atrophy and consequent recession of the naturally concave surface of the bodies allow the edges of the discs covered by the bony layer to form prominent projections (Birmingham

FIG. 307.—Exuberant ossification of the anterior common ligament over the cervical and upper dorsal vertebræ of a lion. Front view (*No.* 2456, *R.C.S. Museum*).

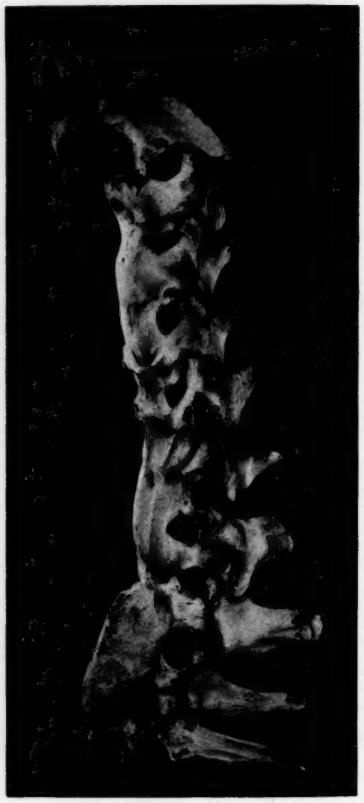


FIG. 308.—Lateral view of the same specimen as shown in *Fig.* 307.

University Pathological Museum, No. 93 (0.47) 85.2a and 2b). There is no alteration of the articular surfaces of the bodies, which are sharply demarcated from the new osseous layer.

Among the ancient Nubian bones in the R.C.S. Museum are several specimens illustrating degrees of spondylitis ossificans ligamentosa. In No. 205 ossification is limited almost entirely to the anterior common ligament over several dorsal vertebræ. In No. 206 ossification extends on to the right lateral surface of the vertebræ, and the laminæ also are fused. In another

(also labelled 205, and much battered) ossification involves the front and lateral surfaces of the bodies, and there is bony ankylosis of the articular processes and ossification of the supraspinous ligament.

Ossification of the anterior common ligament is obviously only a stage, and probably an early one, in a morbid process which is prone to involve other ligaments and to merge into the pronounced form of the disease. But even in the well-marked affection, curiously, it may be completely absent (*see Cases* 2 and 3). It is, however, quite common in certain animals—more particularly in horses and asses. The following account of it is given in Youatt and Watson's book on the horse (4th ed., 249) :—

" If the horse be much overweighted, or violently exercised or suddenly pulled upon its haunches, these ligaments are strained. Inflammation follows. The ligaments are changed into bone, and the joints of the back lose their springiness and ease of motion—or rather, in point of fact, they cease to exist.

" On account of the too hard service required from them, and especially before they have gained their full strength, there are few old horses who have not some of the bones of the back or loins ankylosed. When this exists to any considerable extent the horse is not pleasant to ride ; he turns with difficulty in his stall, finds it hard, when down, to rise again, and he has a singular straddling action."

Several specimens in the R.C.S. Museum (Nos. 2053, 2054, 2055) illustrate this condition. (*Figs.* 306, 307, 308.)

Another remarkable appearance is seen on sagittal section of these specimens, whether human or equine—namely, the spaces left between the ankylosed vertebræ by the removal of the intervertebral substances during maceration. They may be almost linear, suggestive of atrophic fibrocartilages, but more commonly they appear to have been occupied by discs of normal thickness. Occasionally, too, there are signs of ossification having taken place in the disc itself. This occurs at first in the peripheral parts of it, but it may be more extensive in advanced disease, when the space may be partially obliterated by new bony deposit.

GENERALIZED OSSIFICATION OF THE VERTEBRAL LIGAMENTS.

Morbid Anatomy.—The ligaments usually affected are the anterior common ligament, the posterior common ligament (much less frequently), the ligamenta subflava, the capsular ligaments of the articular processes, the ligaments connecting the ribs with the vertebræ, and the supra- and interspinous ligaments. When the neck is involved, the atlo-axoid and the occipito-atloid articulations may be ankylosed, but the evidence of ligamentous ossification is not so definite here as elsewhere. In well-marked cases the vertebral bodies are overlaid in front and on both sides, and sometimes even behind, by an evenly disposed layer of bone which bridges and conceals the discs. It may extend over a number of bodies, and in some cases from the base of the skull to the front of the sacrum. Its uniformity may be such that its connection with the anterior common ligament might almost be doubted. (*Figs.* 309 and 322).

A sagittal section through such a macerated spine (*Figs.* 310 and 311) shows the superficial disposition of the new bone, and the regular array of intervertebral spaces from which normal-sized discs have been removed. The appearance of the section comes as a surprise, for before division the spine looks like a solid bony cylinder. The discs evidently had been preserved from pressure by the fixation of the vertebræ, and were probably healthy. Ankylosis of the articular processes and of the ribs with the vertebræ is partly

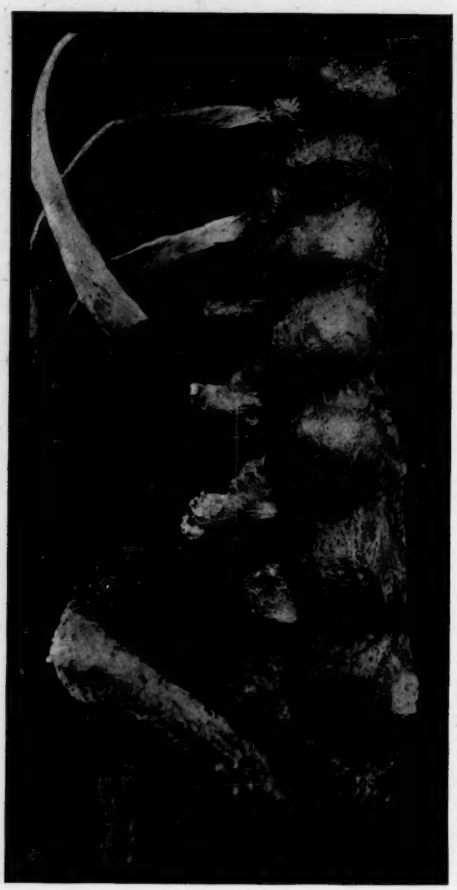


FIG. 309.—Spondylitis ossificans ligamentosa—the generalized form. The plate shows the moniliform thickening corresponding to the vertebral discs, and the atrophic recession of the bodies between the prominences (*No.* 2109, *R.C.S. Museum*).

FIG. 310.—Sagittal section of the spine shown in *Fig.* 309. Note the ossified anterior common ligament, the spaces left by the removal of the discs, and the fusion at the bases of many spines. The groove running down the centre of the bodies is an artefact.

due to the bony change in the ligaments and partly to arthritis, for if a suitable section be made, the joint cavities are seen to be either partially or completely obliterated and the cancellous tissue of the two bones to be continuous (*Fig.* 312). On the other hand, bony union of the articular surfaces of the bodies is delayed, because the discs persist long after vertebral ankylosis has taken place. When it does occur it is due to ossification of the intervertebral substance advancing from the periphery.

This method of invasion of the disc, though a small point, may be added to other facts which tend to show an affinity between spondylitis ossificans ligamentosa and rheumatoid arthritis. The others are: the occurrence in many cases of rheumatoid arthritis in the joints of the extremities, and the frequent presence of an infective focus prior to the onset of the disease.

Mention must not be omitted of those rugosities, tubercles, and stalactitic processes which are often present on the osseous surfaces in the neighbourhood of the joint changes and are obviously abnormal. They may be associated with the attachments of the interspinous and supraspinous ligaments, and as they advance lead to complete ossification of those ligaments. They are also in evidence along the iliac crests and on the front of the pubic bodies and the pubic and ischiatic rami, and in these situations they suggest a connection with the attachment and action of the various muscles. (*Fig.* 313.)

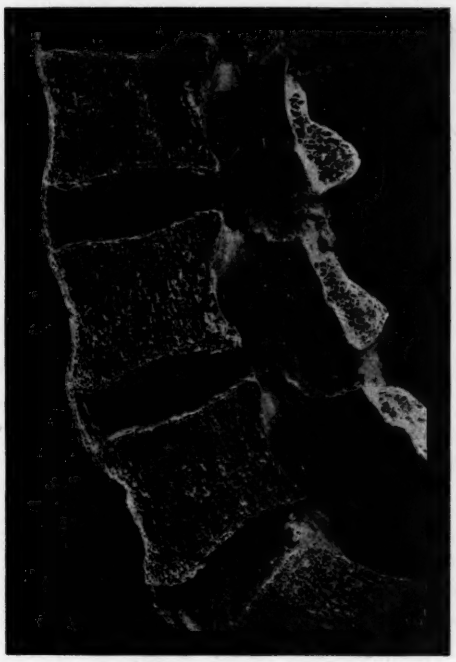


FIG. 311.—The 3rd, 4th, and 5th lumbar vertebræ of the spine shown in *Figs.* 309 and 310, on a larger scale.

Pathogenesis.—In the pathogenesis of the generalized affection the toxic factor is probably supreme, but in

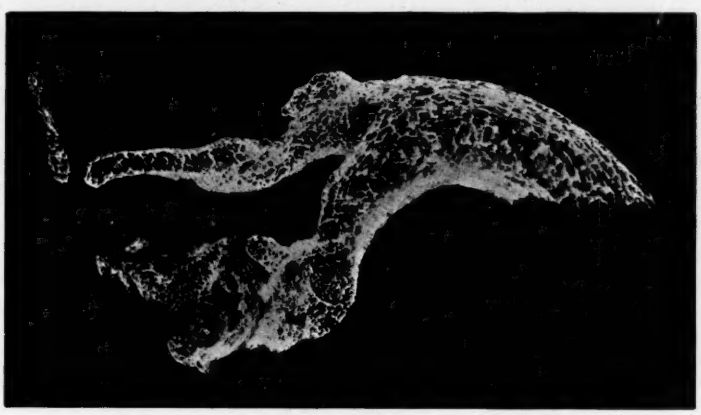


FIG. 312.—Transverse section through the articulation of the 1st rib with the vertebra, showing bony ankylosis. From the same spine as that shown in *Figs.* 309, 310, and 311.

ossification of the anterior common ligament alone, traumatism, in the form of ligamentous strain, is probably more important than toxins.

In this connection, where horses are concerned, it must not be overlooked that pyorrhœa is of frequent occurrence owing to the impaction of particles of food (hay, oats, etc.) between the alveolar border and the gums.

FIG. 313.—Pelvis showing spondylitis ossificans ligamentosa involving the lumbosacral joint. The sacro-iliac synchondrosis and the symphysis pubis are ankylosed. Note the rough osseous nodules in front of the pubic bodies, along the sides of the pubic arch, and at the anterior superior spines (*No.* 4585, *Guy's Hospital Museum*).

CLINICAL VARIETIES.

The clinical picture of the generalized form of spondylitis ossificans ligamentosa is sufficiently distinctive. The variations depend upon the extent to which the vertebral column is invaded, and the way in which the joints of the extremities suffer.

Considerable attention has been devoted to certain cases in which the proximal joints (root joints) of the extremities have shared the ankylosing affection of the spine. They are often alluded to as the Marie-Strümpell type, and Pierre Marie, who suggested the name 'rhizomelic spondylitis', defined the condition as "extremely pronounced rigidity of the spine and ankylosis of the hip- and shoulder-joints". The case described by Strümpell[1] was the following:—

Case 1 (*Fig.* 314).—In a fisherman, age 39, the disease had begun without warning three and a half years previously with pains in the loins, right hip, and thigh. The loin pain sometimes radiated to the stomach region. The pains were never very violent or long-continued, and the stiffness in the loins was more troublesome than the pain. The movements of the head, cervical spine, and upper extremities were normal. The lower dorsal and lumbar spine was rigid and remained like a rod in bending and lateral movements. The upper part of the body was bent forward in standing and walking. The dorsal muscles were atrophic and strangely firm and solid, and the hip-joints were fixed in slight flexion. Under anæsthesia there was no relaxation of the spinal rigidity, and limitation of movement and crepitation were present in the right hip.

He had previously described a more advanced case in his *Lehrbuch*,[2] and had emphasized the progressive course of the disease which "without pain ends in complete ankylosis of the spine and hips, so that the head, trunk, and femurs are firmly fixed to one another and are completely rigid, while all the other joints retain normal mobility", and a quite peculiar modification of the attitude and gait results.

An exhaustive paper on this condition was published by Léri, who was associated with Marie. He referred to the case recorded by Hilton Fagge[3] as the best example of rhizomelic spondylitis that he had met with. The following description of a specimen from that case and the history are taken from the Guy's Hospital Museum Catalogue (No. 4450).

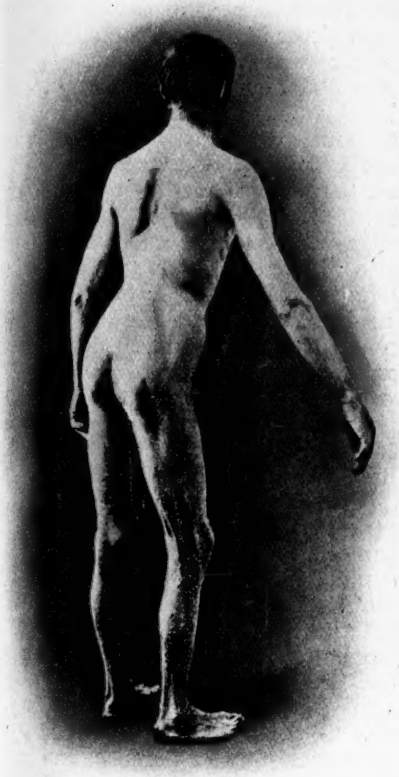


FIG. 314.—*Case* 1. Spondylitis ossificans ligamentosa. Strümpell's case of rhizomelic spondylitis.

Case 2.—George B., age 34, admitted under Dr. Wilks for bronchiectasis, from which he died. He had been a wire worker, and said that although his chest was never well-formed he had always had good health until four years before admission. He then complained of pain in the abdomen, constipation, and loss of appetite. A year ago he began to stoop a great deal, and the abdominal pain returned, so that he was obliged to give up work. At the time he developed a severe cough, and brought up large quantities of sputum. On admission he was unable to walk without assistance, his spine was much bowed, and was completely fixed in the dorsal and lumbar regions, and only capable of a slight degree of movement in the cervical region; his chest was flattened at the sides, and so great was the degree of kyphosis that the abdomen as low as the umbilicus was in contact with the skin covering the lower part of the thorax. At the post-mortem the bronchi of both lungs were found to be widely dilated, but the alveoli appeared to be healthy. There was no sign of tuberculous disease. The heart was hypertrophied, and the great vessels were atheromatous.

The Specimen.—The vertebræ are ankylosed by deposits of bone between the articular processes, laminæ, and spinous processes, and to one of them a portion of rib is similarly united. (Fagge states that the ribs generally were ankylosed to the vertebræ, and on section of one of them there was perfect continuity of one bone with the other.) There is no lipping of the bodies as commonly seen in osteo-arthritis. Their substance is soft (they could be cut with a knife), and both the cancellous tissue and its compact covering have undergone much rarefaction. The hip-joint is the seat of fibrous ankylosis. The line of the articulation as seen on section is natural, and the head of the femur has preserved its normal contour except at its margins, where some erosion has taken place. (*Fig.* 315.)

There are very few post-mortem records of cases of spondylitis deformans, but in the following case of Léri's[4] we have fortunately a second instance of a good clinical history, with the report of the subsequent examination of the spine.

Case 3.—A cook (*Coq*), age 39, was completely cured of a gonorrhœa in 1877. In 1888 he had an acute illness diagnosed as 'acute rheumatism with purpura', and shortly after his recovery, and following some fugitive joint pains, he experienced severe pain in the loins and sacrum, with noises at the top of the sacrum and in the hip. A long rheumatic and varying illness followed, and in 1890 the spine was found immobile. In 1891 the neck became fixed, and the shoulders were attacked. In 1898 his condition was as follows. The cervical spine formed an angle of 140° with the trunk; the chin was close to the sternum; the lower limbs were straight, but flexed at the hips at an angle of 125°. The spine, head, hips, and knees were absolutely rigid, and the movements of the ankles and toes were somewhat affected. Those of the shoulders and lower jaw were impaired, and crepitation was present in the shoulder-joints. The thorax was flattened antero-posteriorly and the abdomen voluminous and spread out. There was no sign of nerve trouble. The patient lay in a half-sitting position, with legs extended, and the upper part of his back and head resting against heaped-up pillows. If his legs were vertical, his head projected a yard in front and caused him to fall. Léri compared his body to a see-saw with the fulcrum at the hips. If his back lay on the bed, his legs went up in the air.

FIG. 315.—*Case* 2. Four vertebræ and a hip-joint from Hilton Fagge's case (*No.* 4450, *Guy's Hospital Museum*).

Examination of the spine after death showed that there was no ossification on the front of the vertebræ: both anterior and posterior common ligaments were unaffected. Ankylosis had occurred between the anterior portions of the lumbar vertebræ for a depth of 2 to 5 mm. between the bodies, beyond which the discs preserved their ordinary biconvex shape, and showed no diminution of thickness. The posterior part of the disc between the 6th and 7th dorsal vertebræ was ossified and united the two bodies. The occiput was united to both lateral masses of the atlas, the anterior arch of the atlas to the odontoid process of the axis, and the lateral masses of the atlas to the articular processes of the axis. The posterior arch of the atlas was wanting.

The articular processes were absolutely ankylosed the whole length of the column. Many laminæ were united from ossification of the ligamenta subflava. The spinous processes were nowhere completely ankylosed. The ribs were ankylosed to the vertebræ by almost all their ligaments, at both costovertebral and costotransverse articulations, and at the latter the two bones fused. The intervertebral foramina were only slightly narrowed, and the soft parts there and in the canal were quite normal and raised no suspicion of inflammation.

Pain and ankylosis are the special features of spondylitis ossificans ligamentosa. The one seems to be the precursor of the other. In the rhizomelic form the pain is apt to be located in the lumbar and sacrococcygeal regions or in the hips. It may occasionally be altogether absent, but in most cases it is continuous, and aggravated by certain positions or on movement. Its onset is sometimes sudden, and may be so violent as to be agonizing. Long-continued fixed pain in the loins or sacral region is very suggestive.

The diagnostic sign is ankylosis. As a rule, it extends from below upwards, involving the lumbar, dorsal, and cervical spine in this order. In the lumbar region it is preceded by a painful period which disappears as ankylosis takes place. There is said to be marked freedom from pain when the dorsal region is attacked, contrasting with the pain which ushers in ankylosis in the lumbar region, and at a later date in the cervical spine. It has been attributed to the normal want of mobility in the dorsal spine. Though extension to the cervical region may be steadily progressive, there is often a considerable interval—even a year or more—before that part of the spine is attacked. Its implication is marked by pains in the neck. The hips become involved when the disease is active in the lumbar region; the shoulder, sternoclavicular, and temporomandibular joints are in danger when the cervical and upper dorsal spine is attacked; and ankylosis of the ribs to the vertebræ probably synchronizes with dorsal mischief; but this condition can only be recognized by the respiration becoming entirely abdominal. Léri allows a certain elasticity to the original conception of rhizomelic spondylitis by admitting that the peripheral joints of both upper and lower extremities may exceptionally be invaded, and that ankylosis in the shoulder-joints is probably uncommon.

Fig. 316.—One of P. Marie's cases showing the bent ankylosed position in rhizomelic spondylitis (*Rev. de méd.*, 1898, xviii, 290).

There is no rule as to the deformity produced by rhizomelic spondylitis (*Figs.* 314, 316). The spine may be greatly curved or remain extended, or, as Léri puts it, 'less flexed'. The lumbar curve is apt to be obliterated, and a forward bend to develop in the upper dorsal region, so that, with the hips ankylosed in flexion, the trunk takes the form of an arc. Probably the deformity is in part determined by an attitude assumed for ease—only so can we explain why in some cases the head is flexed on the spine, and in others ankylosed in complete extension (*Fig.* 317). But kyphosis is likely to develop when rarefaction and softening of the vertebral bodies is pronounced (cf. *Case* 2).

So far the description of the generalized form of spondylitis ossificans ligamentosa has been concerned with the type known as rhizomelic. There are, however, other cases in which the disease is so widespread that practically all the joints of the body become ankylosed. Sir Jonathan Hutchinson[5] has described one in his *Archives*.

Case 4.—The patient, a rather florid man, age 54, had suffered for twenty years, and was almost literally one piece of bone. Knees, ankles, and hips were quite stiff, and so were his elbows, shoulders, and wrists. The last-named joints were still somewhat swollen, and not absolutely ankylosed. His hands and feet were distorted, and all his digits bent into the palm. His hands lay on his chest, and he could not

lift them. His lower jaw was ankylosed, but he could still eat when food was put into his mouth. He attributed his attack to having been put in a damp bed. He had never had acute rheumatism, and his symptoms had developed very gradually. For a long time he helped himself about with sticks ; then for six years he went about on crutches ; then for eight in a bath chair ; and finally for seven he had been bedridden in Lambeth Infirmary. He said that he was never free from aching pain. He had been for many years the subject of stricture and had had gonorrhœa before the rheumatism set in. . . . The whole of the spinal column with the exception of the neck appeared to be stiff.

The following very similar instance came under my own observation.

FIG. 317.—Spondylitis ossificans ligamentosa showing the skull ankylosed in complete extension (*No.* 93.852, *Manchester University Pathological Museum*).

Case 5.—A gentleman, age 37, was the subject of an ankylosis so general and so complete that he could only produce a little movement in two or three toes, and twitch one shoulder slightly. His spine was quite rigid, but his temporo-mandibular joints remained free.

The illness began with painless effusion into both knee-joints just when a subacute gonorrhœa had apparently recovered. The disease ran a rapid course and in three years reduced him to the state I have described. It did not seem to have been attended with much pain, but he had become blind in both eyes from the effects of iritis. Some time before he contracted the gonorrhœa he had suffered from pleuritic effusion, and his knee-joints had filled with fluid during his convalescence ; from this illness he had made a satisfactory recovery.

There can be no doubt of the close relation of these cases to gonorrhœa, and that the completeness of the ankylosis gave rise to a remarkable clinical picture. It is unfortunate that no record of the morbid anatomy of similar cases could be found to compare with that of rhizomelic spondylitis described in *Cases* 2 and 3. But two points about those cases may be impressed: (1) There was no ossification along the front of the vertebral bodies—a fact which is remarkable when we remember the frequency with which such ossification is present in museum specimens ; and (2) There was no associated gonorrhœa in *Case* 2 and one of no significance probably in *Case* 3.

It may be that, as knowledge accumulates, a definite form of spondylitis ossificans ligamentosa will be found to be associated with gonorrhœa as clearly

as Charcot's joint disease is with syphilis, but at the present time this cannot be shown.*

II. CASES CHARACTERIZED BY AN UPPER DORSAL CURVE AND ATROPHY OF THE INTERVERTEBRAL SUBSTANCES.

(*Spondylitis Muscularis.*)

CLINICAL FEATURES.

In this condition the mischief is usually limited to the upper part of the spine, and the joints of the extremities are not involved. Slight degrees of it are familiar to all of us in hump-backed and round-shouldered people; but the more pronounced stages are uncommon, and it is to them that the term spondylitis deformans is more particularly applied.

FIG. 318.—*Case* 6. Spondylitis muscularis. The patient in Marie and Astie's case—' cyphose hérédo-traumatique '.

FIG. 319.—The sister of the patient shown in *Fig.* 318.

Through the kindness of Mr. H. E. Powell.

In many instances the foundation of the mischief is laid in early life, and after a quiescent period of many years the disease may become aggravated or relapse as a consequence of various untoward circumstances. In well-recorded instances the presence of a stoop dating back as long as the patient was able to remember is often mentioned. The most important element in its causation

---

A good clinical case is recorded by M. Paul Claisse, *Bull. Soc. méd. Hôp. de Paris*, 1904, xxi, 3s., 1227.

is the *feeble tone of the muscles*, which forces itself upon the attention more particularly in those which normally maintain the erect position of the upper spine and the balance of the head.

Only slightly less material are the *atrophic changes that occur in the intervertebral discs*. These, however, are secondary to the muscular failure, and are caused by the increase of pressure at certain points consequent upon the forward bending of the spine. Other predisposing factors that may be referred to here are *trauma* and *heredity*. The determination of the slighter degrees of upper dorsal kyphosis is often ascribed to injury, occasionally on good grounds; but in many cases of the graver sort a very definite association with injury exists. Sometimes—as P. Marie and Astie[6] have pointed out—a history of curvature in other members of a family can be obtained, and they distinguished cases in which hereditary tendency was combined with undoubted traumatism by the title 'cyphose hérédo-traumatique'. The case published by them is as follows:—

Case 6.—A man, age 53, who, some days after a fall on his back in which the spine had impinged on a metal utensil he had been carrying, experienced violent pain, a sensation of extreme weight, and—a peculiarly interesting fact—a considerable bending of the vertebral column forwards. He came of a family in which there was a tendency to curvature. His father was bent. His sister, also, notably so. He himself was bent before the accident, but the extraordinary kyphosis actually present developed in the first week following the injury. Careful inquiries were made from the patient and relatives, and there could be no doubt of the direct relation between the actual kyphosis and the trauma. The photographs of the patient and his sister (*Figs.* 318, 319) published with Marie and Astie's paper show very similar pronounced upper dorsal curvatures.

A similar hereditary factor was present in the following instance on the maternal side.

Case 7.—This case was that of a gentleman, age 73, in whom a severe upper kyphotic curve began to develop in boyhood, after a fall down a flight of stairs in which he hurt his back and broke a clavicle. Stationary for the greater part of his life, it increased so rapidly with failing health that his height diminished 7 inches in a few years. The deterioration in his general condition was initiated by a severe and progressive arteriosclerosis. (Personal observation.) *

The explanation of the sudden increase of the curvature in *Case* 6 is probably compression and collapse of the rarefied cancellous tissue of the bodies, whilst that of the gradual loss of height in *Case* 7 may be based partly upon compression of rarefied bone and partly on accelerated atrophy of the discs.

The subjects of spondylitis muscularis are usually conscious of a feeling of weakness in the back and of aching pain in the region of the curvature, especially after exercise. In some cases complaint is made of pain in the area of distribution of the spinal nerves involved. This has been suspected

---

* Messrs. Danlos, Apert, and Levy Frankel[7] recorded a remarkable instance of a familial tendency to pronounced upper dorsal kyphosis appearing in all its subjects between 5 and 7 years of age. It was associated in one instance with some degree of acromegalic gigantism, and in others with prominent jaws and tall stature. The probability of a traumatic factor was absent in most of them. The possibility of the curvature being a pituitary stigma was considered, but the evidence in support of it was not convincing.

to depend upon compression of the nerve-roots in their passage through the intervertebral foramina, but in the very few observations that have been made this has not been found. Garrod[8] is of opinion that these nerve-root symptoms " deserve a more prominent place in the symptomatology of the disease* than is usually given in published descriptions ", and states that patients in ignorance of their rigid spines will sometimes apply for the relief of radiating pains, which may be of a very distressing character.

Upper spinal kyphosis associated with nerve-root symptoms has been carefully studied by Von Bechterew,[9] of Petrograd. In a series of papers he recorded a number of cases in which, in addition to rigidity and a kyphotic bend 'above the mammary region', symptoms were present indicative of degenerative changes in the corresponding nerve-roots. These symptoms included: (1) A paretic condition of the muscles of the trunk, neck, and extremities, with slight atrophy of the back and scapular muscles; (2) A diminished sensibility, chiefly in the cutaneous branches of the dorsal and lower cervical nerves, and sometimes of the lumbar nerves, with other variable objective and subjective symptoms; (3) In some cases evidence of motor-nerve irritation, such as twitching, contracture, etc.

Unfortunately he was unable to check his observations by post-mortem evidence until 1899, when a case he had already recorded[10] died of basal pneumonia, and provided the following pathological results:—

An upper dorsal kyphotic curve was present without compensatory lordosis in the lumbar region. The curve was absolutely immobile, and the bodies of the vertebræ forming it were fused anteriorly; the intervertebral discs were either atrophic or had completely disappeared. Elsewhere the discs were normal and the mobility of the lower dorsal and lumbar regions was only slightly restricted. Illustrations show that the kyphosis was not severe and that the various vertebral ligaments were not ossified.

The examination of the nervous system showed the cranial dura and pia to be hyperæmic; the cervical spinal dura to be rather thickened but everywhere smooth. Where the roots had penetrated the dura, the spinal ganglia were to some extent adherent; but it was uncertain if this adhesion was pathological. The dorsal nerves were of a light-grey colour as the result of degeneration, but the lumbar and sacral nerves showed no evidence of degeneration to the naked eye. Of the roots, the cervical were normal in colour, the lumbar and upper dorsal partly grey and partly white. The spinal cord was moderately soft, especially in the lower dorsal region, and the grey matter was hyperæmic in the cervical and dorsal portions.

The microscopical changes were most striking in the lower cervical and upper dorsal region, and less so in the lower dorsal and lumbar enlargement. There were no changes in the upper cervical region.

In the first-named regions—especially in the upper dorsal—the posterior roots were almost entirely degenerated throughout, but the anterior roots showed a less intense fibro-degeneration. In the cord evidence of degeneration was most pronounced in the upper dorsal and lower cervical parts—the region of maximum root affection. The columns of Goll and of Burdach

* He is here referring to spondylitis deformans generally.

were the parts affected, and in the latter the degeneration corresponded to the entry of the posterior roots.

The degeneration in Goll's column began at the level of the 7th dorsal nerve-root and reached its maximum in the cervical part of the cord. A diffuse degeneration was present in the ventral columns in the upper dorsal and lower cervical cord and in the adjoining part of the lateral columns, also in the peripheral part of the cord in the region of the anterior roots. This latter was most marked in the cervical cord, and was apparently related to the nerve-roots themselves. The former corresponded to a system of sensory paths which cross in the anterior commissure.

The grey matter of the cord showed no changes, but in the spinal-root ganglia many cells were degenerated and atrophied and showed leucocytes in their interior. There was slight degeneration of the peripheral nerves, which was continuous with that of the roots. The atrophied muscles showed loss of cross-striation and fatty degeneration of the muscular fibres.[11]

On these findings Von Bechterew suggested that the degeneration of the posterior roots was to be explained by the chronic (inflammatory) process in the meninges which resulted in compression of the roots at their point of entry into the cord, and in the adhesion of the spinal ganglia to the dura mater and the surrounding connective tissue. The meningeal mischief was therefore the primary condition, and the degeneration of the spinal nerves secondary to it. The nerve degeneration led to paresis of the muscles and inability to maintain the erect posture and the balance of the head, and these in their turn were responsible for the kyphosis. The abdominal respiration was to be attributed to the paretic state of the intercostals. Finally, the kyphosis caused compression and atrophy of the discs until the bone surfaces came in contact anteriorly, when bony union followed.

Much interest attaches to this post-mortem corroboration of Von Bechterew's observations, and to his conclusions. The latter, in view of the uncertainty as to the pathological adhesion of the ganglia to the dura, would seem to rest upon a somewhat insecure basis, and further investigations are desirable before the pathology of this type of spondylitis muscularis can be considered to be settled.

From Goldthwait[12] we get the impression that root symptoms are due to the pressure of inflammatory swelling at the intervertebral foramina resulting from the disease in the region of the transverse processes, and that, as we might expect if that is so, the symptoms usually improve, though in many instances recovery is never complete. He also mentions that a pressure paralysis very similar to that seen in Pott's disease may result from osseous deposit in the posterior ligament, and that relief of the symptoms usually takes place.

SPONDYLITIS SENILIS.

The spinal changes associated with old age may be regarded as a variation of spondylitis muscularis. Weakness and atrophy of the muscles progress simultaneously with the shrinkage and disappearance of the intervertebral substances. Probably both result from degenerative changes in the cardio-vascular system. The disappearance of the discs alone is sufficient to produce

a marked dorsal kyphosis, but as, during life, the mechanical situation steadily alters for the worse from this cause, the failure of muscular power that accompanies it will aggravate the deformity. The following extract from Morris's *Anatomy of the Joints* (p. 70) will help these senile changes to be understood.

"The intervertebral substances vary in shape with the bodies of the vertebræ which they unite. They have therefore a wider area the lower they are situated in the column. Their thickness is also greater in the lumbar region. Together they form nearly one quarter of the whole length of the column—so that in a column of 28 inches, the intervertebral substances together measure about 7 inches. Their total length along their front aspect is greater than that along their posterior surface, and this difference is chiefly due to the cervical and lumbar substances. Thus they are the cause of the convexity forwards in the cervical region, and increase that in the lumbar.

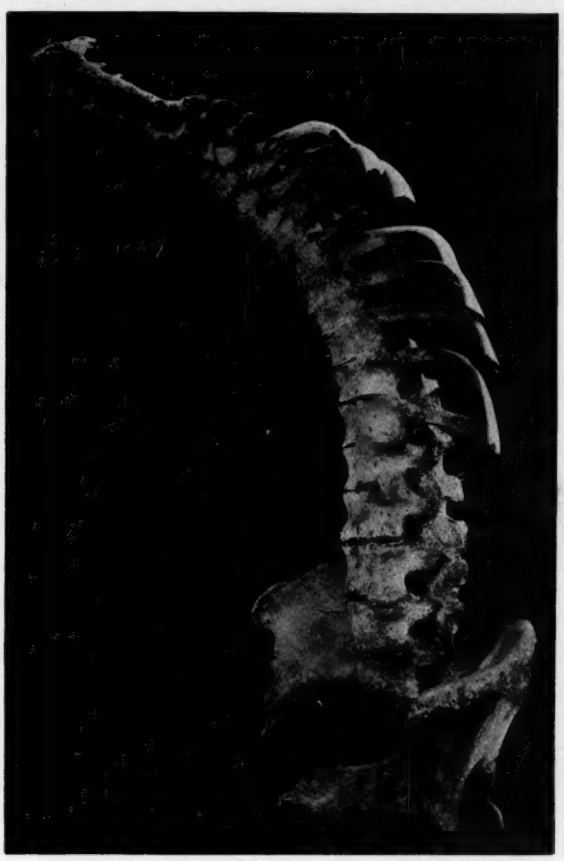


FIG. 320.—Another example of spondylitis ossificans ligamentosa, showing ossification in front of the bodies, bony ankylosis of the articular processes and of the ribs to the vertebræ (*No.* 2050, *R.C.S. Museum*).

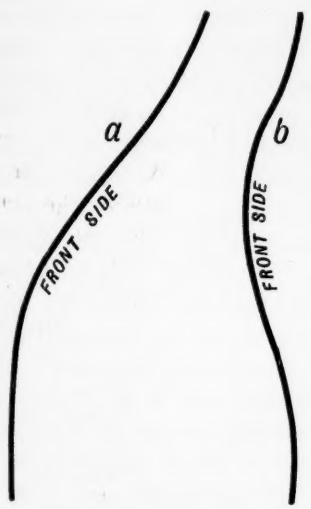


FIG. 321.—Diagram after Morris.

It will be seen on referring to *Fig.* 321 how great is the difference in the curve of the spine (*b*) in the recent state, and (*a*) in the dry, after the intervertebral substances have been removed and the vertebræ are placed in close contact with each other, both with respect to their bodies and articular processes. In the dry state the convexity of the cervical and lumbar regions almost disappears, and the vertebræ present one great curve the concavity of which looks forward and is most marked a little below the middle of the dorsal region. Such is the curve of old age, owing to the shrinking and drying up of the intervertebral substances, which in their perfect state not only straighten out the column, but give to it the forward projections in the cervical and lumbar regions, and produce those gentle curves which have led in all ages to its being called 'the line of beauty'. The concavity forwards

of the dorsal region is almost entirely due to the shape of the bodies of the vertebræ, but it is somewhat increased by the intervertebral substances."

Museum specimens of uncomplicated senile changes in the spine are astonishingly rare. In the R.C.S. Museum (No. 2108, Hunterian specimen—*Fig.* 322) is a preparation which is believed to be the result of old age. It shows a posterior curve of the upper dorsal spine. The curve amounts almost to a right angle and involves the dorsal vertebræ from the 2nd to the 7th. Nearly all the dorsal intervertebral substances are obliterated or extremely narrow. The anterior parts of the articular surfaces of the bodies from the 3rd to the 7th appear to be fused. There is no ossification of the ligaments.

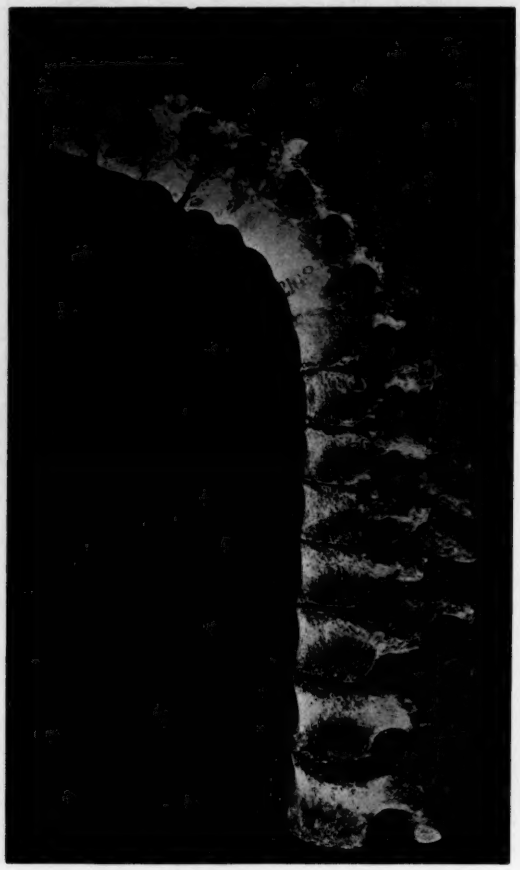


FIG. 322.—Spondylitis (muscularis) senilis (*No.* 2108, *R.C.S. Museum*).

In the Birmingham University Pathological Museum is another (No. 93 (04.7) 85.2), probably of a similar nature. It shows a kyphotic curve of the dorsal spine to a right angle having the 7th and 8th dorsal vertebræ at its summit. The intervertebral substances (both lumbar and dorsal) have to a great extent been absorbed, and there is fusion of the bodies from the 4th to the 10th dorsal vertebræ. A thin layer of bone (anterior common ligament) is spread over the anterior surface of the concavity of the curve and is limited to the vertebræ mentioned, and there is slight ossification also of the ligaments between the 7th, 8th, and 9th spines.

In neither of these specimens is there sufficient evidence of osteo-arthritis to suspect that disease as the cause of the kyphosis, and in neither unfortunately are there any clinical notes.

PATHOGENESIS.

Abundant evidence points to the toxic origin of spondylitis ossificans ligamentosa. The toxins are usually derived from some infective focus. In many cases, though not in all, this is of a septic nature, and frequently it is gonorrhœal. The connection is often so obvious that it cannot now be overlooked as it used to be in the days when micro-organisms and toxins were unknown.

In spondylitis muscularis, however, no such direct association is manifest, although in few cases can infection have been completely absent at all times throughout the development and duration of the disease. The muscular weakness which is the primary factor in the early stage may, it is true, be the consequence of some illness due to an infection, but only in this indirect way can the influence of toxins other than metabolic toxins be suspected.

The rôle which muscular weakness, atrophy of discs, trauma, heredity, and old age play in the evolution of the affection has already been considered in the foregoing account; but in particular cases other factors may be present. These would include an occupation entailing a stooping posture or the bearing of burdens upon the upper part of the spine; also undue rapidity of growth or great height, in both of which the muscular development is apt to be inadequate. Prolonged or late rickets obviously, or any debilitating illness, especially in early youth or adolescence, may predispose to this form of kyphosis; so also may myopia or a studious habit, and possibly even a morose temperament, especially if conjoined with a disinclination to a healthy outdoor athletic mode of life.

MORBID ANATOMY.

A comprehensive description of the morbid anatomy is difficult, because there are so few specimens and records on which it can be based. Its chief features, which have already been described in the accounts of the autopsy on Von Bechterew's case and museum specimens, are:—

1. A kyphotic curve in the upper or middle dorsal region, arising primarily from muscular weakness.
2. Pressure atrophy of the discs included in the curve, more particularly at their anterior parts.
3. This is followed by fusion of the anterior parts of the articular surfaces of the bodies, when, owing to the disappearance of the intervening portions of the discs, the bony surfaces of adjacent vertebræ are brought into contact.
4. Often a slight degree of lateral bending is present at the upper part of the curve, and on the concavity of this bend the bodies may be slightly compressed, the disc edges may have disappeared, and fusion of adjacent vertebræ taken place.
5. Owing to moulding of the rarefied cancellous tissue resulting from the combination of curves, the bodies may become altered in shape; the side-to-side diameter may be diminished and the antero-posterior slightly increased (*Fig.* 322). The compact tissue of the concavity may be strengthened by sclerosis. A similar need for increased strength was probably responsible for the purely local ossification of the anterior common ligament on the concavity of the curve in the Birmingham specimen described in the section on senile changes.
6. Lastly, in the senile form, atrophy of the discs is not limited to the region of the curve, but extends throughout the spine.

When spondylitis ossificans ligamentosa is compared with spondylitis muscularis, it seems remarkable that they could ever have been regarded as

simply different types of the same disease. Not only are the signs and morbid anatomy of the former quite different from those of the latter, but in each condition they are distinctive. The pathogenesis also is not the same.

The confused condition of our knowledge of spondylitis deformans arises not only from the absence of clinical notes from museum specimens, but also from the fact that the literature is largely composed of records of cases published during their lifetime. It is quite rare to come across a fully reported case with the results of a pathological examination attached. There can, however, be no doubt that spondylitis ossificans ligamentosa and spondylitis muscularis are distinct affections, and should be regarded as separate diseases.

III. CASES IN WHICH THE CHANGES ARE OF AN OSTEO-ARTHRITIC CHARACTER.

(*Spondylitis Osteo-arthritica.*)

Osteo-arthritis of the spine resembles the same disease in the joints of the extremities, but it presents certain remarkable differences. Those characteristic signs—the disintegration and removal of articular and interarticular cartilages, and the formation of marginal osteophytes—are as pronounced in the spinal joints as elsewhere. On the other hand, an absence of eburnation and some tendency to ankylosis are peculiar to the spinal affection.

It is easy to explain the absence of eburnation, for the amphi-arthrodial joints connecting the vertebral bodies do not permit of such movement as is necessary for its production. The only joints between the articular processes (arthrodial) in which movement is tolerably free are the lateral atlo-axoid. There is free mobility also in the occipito-atloid joints (ginglymo-arthrodial). It is in these joints that eburnation might be expected to occur, and the only articulations in which I have found it well marked were an atlo-axoid (*see Specimen* 6 *infra*) and one between the 3rd and 4th cervical vertebræ in a case where the axis and 3rd cervical were fused (*Specimen* 7). Bony ankylosis of the spine in osteo-arthritis is not so frequent as it is believed to be. In museums examples of spondylitis ossificans ligamentosa largely predominate over those of spondylitis osteo-arthritica, and as these are very commonly described as osteo-arthritis, confusion naturally results.

A study of those specimens which are unmistakably the result of osteo-arthritis, and in which the diagnosis was clinched, with reasonable frequency for museum specimens, by a record of osteo-arthritis in other joints, showed that fusion of the articular surfaces of the bodies and of the articular processes was unusual, that ankylosis of the ribs to the vertebræ had not occurred, and that the laminæ and spinous processes were not united by bone. When bony ankylosis was present it was commonly due to the fusion of the osteophytic lipping of adjacent vertebræ, but more often, even in advanced cases, the line of contact persisted without fusion. Fusion of the vertebral bodies did occasionally occur, but it was under circumstances in which pressure and probably complete immobility were evidently determining factors. It was noticeable that the joints in which the atlas participated were liable to

bony ankylosis when attacked, and when ankylosed exhibited no sign of osteophytes.*

A study of the following descriptions will show the justification for these statements.

Specimen 1.—The lower dorsal, lumbar, and sacral spine of an aged female (No. 2048, R.C.S. Museum: Sir J. E. Erichsen) shows marked osteo-arthritic changes (*Fig.* 323). The discs must have been destroyed in all the lumbar joints except the lumbar sacral. Contiguous articular surfaces of the bodies of the 1st and 2nd lumbar vertebræ and also of the 4th and 5th present complete areas of uneven rarefied bone whose inequalities appear almost as if adapted to fit together. There is no ankylosis of any of the vertebræ, though the outspread marginal lippings of adjacent vertebræ have evidently been in contact in many places.

Two other specimens from the same patient illustrate osteo-arthritis of other joints: No. 1989 is a left shoulder-joint, and No. 1905 is a beautiful example of advanced osteo-arthritis of the knee. It is surprising that fusion of the vertebral bodies had not taken place in this case. Fibrous tissue must certainly have united the 1st and 2nd, and the 4th and 5th lumbar vertebræ, and ossification was probably advancing into it and across it to convert a fibrous into a bony ankylosis; but the ossifying process was evidently very slow, and the new trabecula had not consolidated sufficiently to withstand the effects of maceration.

---

* In a series of papers of exceptional interest Arbuthnot Lane[13] advanced the view that "pressure or force applied in one form or another is the chief if not the sole factor" in the production of the changes in osteo-arthritis. In developing his thesis Lane pointed out that "the vertebral column and the joints connected with it reacted to pressure in three different ways according to the manner it is exerted upon the bones and joints".

1. The changes due to *direct transmission of pressure* constitute the first group. They are:—

a. Bones are rendered more dense and strong; this is most marked at points of greatest strain.

b. Intervening soft structures are removed—namely, interarticular fibrocartilage, articular cartilage, interspinous ligaments, ligamenta subflava.

c. With the removal of the cartilage, the subjacent bone becomes dense, and marginal lipping takes place. Adjoining vertebræ may be partially synostosed by fusion of these bony outgrowths.

d. On complete removal of the intervening soft parts, fusion of the vertebral bodies may take place, also fusion of the laminæ, the spines and the articular processes, the sacro-iliac synchondroses and symphysis pubis.

2. The second group of changes depends upon *great tension of the ligaments*, a condition the reverse of transmission of pressure.

The spinous processes increase in density and size, encroaching upon the normal area of the ligaments connecting them. They may even become united by bone. These effects are seen in an antero-posterior curve that has to support much weight. When the spine is over-extended ossification of the anterior common ligament may ensue.

3. The third group of changes is brought about by *an oblique incidence of pressure*: as when an antero-posterior upper dorsal curve is produced by carrying heavy loads upon the upper part of the head and neck. Destruction of the cartilages and displacement of the bodies of the vertebræ are prevented by abundant callus which joins the margins of adjacent vertebræ and bridges the cartilages. These show but slight pressure change.

Lane supported his argument by referring to the fusion of the lumbar vertebræ seen in the extinct giant sloth (R.C.S. Museum), attributable, as he believed, to the great weight that was transmitted through them. A similar fusion, probably traceable to a like cause, occurs in the cervical vertebræ of the whale, the porpoise, and the armadillo, and also in the marsupial mole of Australia, a burrowing animal which also shows a horny shield on its snout. In the ordinary field mole there is no fusion of cervical vertebræ and no horny plate. Both moles, however, use their feet in burrowing.

Whatever opinions may exist as to the correctness of Lane's original conception, there can be no question that we owe to him the full recognition of the importance of pressure, and tension of ligaments, in the production of osteo-arthritic changes. It is also fair to note that in a later work he expressed an opinion as to the toxic factor in the causation of osteo-arthritis which is in harmony with the trend of thought at the present day.

Specimen 2.—One of John Hunter's specimens (No. 2049, R.C.S. Museum) shows lipping of most of the vertebral bodies, particularly of those in the lumbar region. The costovertebral articulations have also suffered, and some of the laminae in the dorsal region are affected. There is no ankylosis of the ribs, or of the articular processes, and no fusion of the vertebral bodies, but union has occurred in a few places where adjacent osteophytic protrusions have come into contact.

FIG. 323.—Spondylitis osteo-arthritica. The bodies are held apart by nuts upon an iron rod (*No. 2048, R.C.S. Museum*).

Specimen 3.—A third specimen (No. 2107, R.C.S. Museum: Robert Liston), said to be the spine of an old woman, shows marked lipping in the lumbar region, where there is no ankylosis. Several dorsal vertebræ are united by fusion of osteophytes, but only the bodies of the 4th and 5th dorsal vertebræ are ankylosed by fusion of their articular surfaces. These two vertebræ are hardly lipped at all, and are situated just above the most prominent part of a long dorsal kyphosis. The peculiarity of the curvature rather suggests that the changes of old age are added to those of osteo-arthritis.

Specimen 4.—In another case (No. 1085, St. Bart.'s Hosp. Museum) several dorsal vertebræ are united by ossification of the anterior common ligament. There is marked lipping in the dorsolumbar region, but no fusion of the articular surfaces of the bodies and no ankylosis of the ribs or articular processes. The patient's left knee had been excised for rheumatoid arthritis. (In all probability this means osteo-arthritis.)

Specimen 5.—Marginal osteophytes arise from all the lumbar vertebræ in No. 1085a, St. Bart.'s Hosp. Museum, and the 2nd, 3rd, and 4th are united by a mass of hard bone at the part where the psoas muscle is attached to them.

Specimen 6.—There is in the Manchester University Museum a specimen of unusual interest (No. 93. 85. 2). It is an occipital bone with which the atlas is fused. The left inferior articular surface of the latter bone—on the under surface of its lateral mass—is so worn down that none of its original surface remains. It is surmounted by a rough flange of osteophytic deposit, and is marked by parallel ridges of eburnated bone corresponding in direction with the revolving movement of the joint. But besides this, the atlas is greatly atrophied and the portion of its posterior arch (nearly half) adjoining the osteo-arthritic articulating surface has disappeared. (Cf. *Case* 3.)

The disappearance just referred to is doubtless the result of pressure, partly from the lateral canting of the head and atlas that must have been present, and partly also from the osteophytic outgrowth. The atlo-occipital joints and all that remains of the atlas are firmly synostosed, but there is no lipping at these joints.

In this specimen, in close relation, are three joints greatly changed. In one the changes are diagnostic of osteo-arthritis; in the other two there is ankylosis without the accepted signs of osteo-arthritis, but associated with other changes evidently the result of pressure and partly of disuse. Clearly we must assume that the arthritis is of the same nature in all three cases,

i.e., osteo-arthritic, and that the presence or absence of movement determines the different appearances. It is an illustration of Arbuthnot Lane's aphorism that pressure, after removal of cartilages, will cause fusion if there is no restitution of movement. The fusion of the two dorsal vertebræ in Liston's specimen (*Specimen* 3) is similarly explainable.

It might be suggested that the osteo-arthritic joint was the secondary effect of a congenital synostosis. It is unfortunate that there should be no information as to the condition of the rest of the spine or the other joints, either in this or the next case, but in the light of *Case* 3 (p. 532) I do not think this theory can be seriously entertained.

Specimen 7.—A somewhat similar specimen to the preceding one is No. 2021D in the R.C.S. Museum (*Fig.* 324). In this it is the inferior articular facet of the 3rd cervical vertebra on the left side which presents the ordinary signs of osteo-arthritis, being deeply worn, eburnated and pitted, and widely lipped with new bone, whilst the axis and the 3rd cervical vertebra are synostosed—joints, bodies, and arches—a condition attributed in the catalogue to congenital defect. The specimen was from a heavy woman of 45 who died after a fall downstairs in consequence of a fracture-dislocation (through the odontoid process) between the atlas and axis, with tearing across of the left vertebral artery.

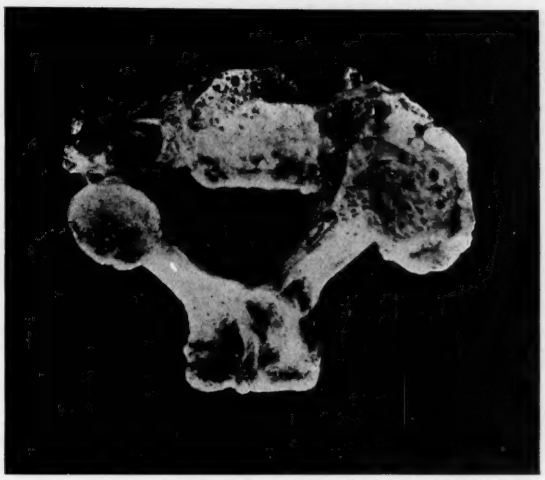


FIG. 324.—Spondylitis osteo-arthritica. Notice the encroachment of the lipping of the body, and also of that of the articular process upon the upper half of the left neural foramen, which might have caused nerve-root symptoms (*No.* 2021*D*, *R.C.S. Museum*).

False Lipping.—Bony protrusions corresponding in position to the intervertebral substances are not necessarily marginal osteophytes or evidence of osteo-arthritis. A moniliform row of such prominences is a frequent feature in spondylitis ossificans ligamentosa and is often regarded as pointing to osteo-arthritis. The abnormality is produced by atrophic changes in the vertebral bodies, and has already been referred to.

Specimen 8.—There are two specimens in the Birmingham University Museum which are significant (Nos. 93 (04.7), 85, 2a and 2b). Together they complete six vertebræ—part of a dorso-lumbar curve which has been divided sagittally. Ankylosis has resulted from ossification of the anterior common ligament and portions of the discs in places. On the left side a vertical row of bosses covers the edges of the discs and the adjacent portions of the sides of adjoining vertebræ, suggesting lipping beneath the lateral margin of the ligament.

On careful investigation it is seen that the phenomenon is produced by atrophic shrinkage of the bodies on that side, deepening the natural concavity of their surfaces and throwing the normal elevations due to the edges of the discs into marked prominence.

The right halves of the bodies retain their normal breadth and shape, and are a great contrast to the contracted left halves. In the lower part of the specimen the protuberances are less marked and the atrophy not so excessive.

Specimen 9.—In the R.C.S. Museum there is a well-known specimen (No. 2105A) which may explain the nature of the 'lipping' so often noticed on the concave surfaces of curves in scoliosis. It shows a lateral lumbar curve of exceptional severity with very prominent protrusions on the concave surface between the bodies. A side-to-side section has been made, and both vertebræ and discs are seen to have acquired a wedge-shaped form with their bases at the convexity of the curve. The apices of the discs show degeneration and atrophy, and in company with the adjoining cancellous tissue on each side, have been elongated outwards on the concave surface, until deep gulfs have been produced in the sides of the bodies between these promontories. Except for this moulding by long-continued pressure, the bone on the face of the section appears only closer where it has been compressed, and shows none of the irregularity which is caused by marginal osteophytes.

I am greatly indebted to the kindness of the curators of the various museums from which I have been permitted to draw much of the material on which this article is based, and also to several friends, particularly Dr. L. J. Kidd and Mr. Plarr, for their willing help in extricating me from the pitfalls of German translation. To all of them I am very grateful.

REFERENCES.

[1] STRÜMPELL, *Deut. Zeits. f. Nervenheilk.*, 1897, xi, 338.
[2] STRÜMPELL, *Lehrbuch*, 1884, xi, 2s., 152.
[3] FAGGE, HILTON, *Trans. Pathol. Soc. Lond.*, xxviii, 201.
[4] LÉRI, *Rev. de Méd.*, 1899, xix, 597.
[5] HUTCHINSON, SIR JONATHAN, *Archives*, iv, 66.
[6] MAIRE, P., and ASTIE, *Presse méd.*, 1897, Oct. 6, No. 8, 205.
[7] DANLOS, APERT, and FRANKEL, *Bull. Soc. méd. Hôp. de Paris*, 1909, xxvii, 3s., 653.
[8] GARROD, Allbutt and Rolleston's *System of Medicine*, iii, 39.
[9] VON BECHTEREW, *Deut. Zeits. f. Nervenh.*, 1897, xi, 327.
[10] VON BECHTEREW, *Neurol. Vestnik* (*Russich*), 1897, v, Hft. 1.
[11] VON BECHTEREW, *Deut. Zeits. f. Nervenh.*, 1899, xv, 52.
[12] GOLDTHWAIT, *Boston Med. and Surg. Jour.*, 1899, cxli, 128.
[13] LANE, ARBUTHNOT, *Trans. Pathol. Soc. Lond.*, xxxv, xxxvi, xxxvii ; *Guy's Hosp. Rep.*, 1886.

SUBPERITONEAL EFFUSIONS SIMULATING ACUTE INTRAPERITONEAL DISEASE.

By J. L. JOYCE, Reading.

The simulation of acute intraperitoneal disease by certain extraperitoneal disorders has long been recognized by surgeons, but writers on acute abdominal disease do not seem to have noticed that this simulation may be produced in a striking manner by a rapid effusion of blood, pus, or serum into the subperitoneal tissues.

My attention was first attracted to this subject by a disaster. I had been asked to remove a fibromyoma of the uterus. The operation was to have been at 8.0 a.m. on the day arranged in a small nursing home, but on the previous evening I was summoned to the home about 11.0 o'clock because the patient had been seized a few hours earlier with acute abdominal pain and vomiting. On my arrival I was told that the patient had begun to complain of severe pain in the lower part of her abdomen during the administration of a soap and water enema at 7.0 p.m., and that she had twice vomited. The patient maintained that the enema was the cause of her illness, and so insisted on this that the nurse who had given the enema, and who had retired to bed, was fetched from an adjoining house to give her version of what had occurred. The nurse stated that she had given the enema with a Higginson's syringe, and that she had passed its bone nozzle through the anus without difficulty. The patient had complained of severe pain in the abdomen after the injection of two or three ounces of the soapy water. The nozzle was withdrawn, and a little blood the nurse had seen on it she attributed to some bleeding piles which were known to be present. After waiting a few minutes, the nurse completed the administration of the enema, and the patient returned the injection with some flatus but without relief of her pain.

The patient had an anxious facial appearance, and was lying on her back with her knees flexed. The temperature was 97° and the pulse-rate 112. The lower part of the abdominal wall moved poorly on respiration and was tender and rigid. On vaginal examination a rounded swelling was found in Douglas's pouch, but the tenderness and rigidity made bimanual examination difficult, and the nature of the swelling could not be determined. Rectal examination was painful; the swelling in Douglas's pouch was felt, but nothing more about its nature was learnt. Some prolapsed piles were seen at the anus, but they were not bleeding.

A medical colleague, in consultation, agreed that some acute intraperitoneal disease was present, probably a twisted ovarian cyst, and that an emergency operation should be performed.

The abdomen was explored through a paramedian incision; the subperitoneal tissues were œdematous; a pedunculated fibroid of the uterus

was the swelling which had been felt in Douglas's pouch, but no intraperitoneal lesion was found which could account for her acute symptoms. Leaving a small drain down to the peritoneum at the lower end of the incision, the abdomen was closed. Next morning the patient was desperately ill; by the evening she was dead.

A limited post-mortem examination was made. The subperitoneal tissues of the pelvis were grossly infiltrated with a seropurulent effusion. A rosette-shaped mass of œdematous piles, which had prolapsed from the right side of the anal canal, projected from the anus and covered its orifice. A hole in the centre of the piles led to a track which ran upwards and forwards into the pelvic tissues outside the rectum. As the examination proceeded, an escape of soapy water through the track explained what had happened: the nurse, mistaking the centre of the rosette-shaped mass of piles for the anal orifice, had pushed the nozzle of the enema through that and injected a portion of the soap and water into the loose cellular tissues outside the rectum. The rapid effusion of inflammatory serum into the subperitoneal tissues of the pelvis, which followed the injury, gave rise to symptoms and signs closely resembling those of an acute intraperitoneal disease.

A similar accident recorded by Fletcher Shaw[1] was not fatal, but resulted in occlusion of the lower part of the rectum. For many years nurses trained in Reading have been taught to give enemata from funnels through soft rubber catheters: a practice worthy of universal adoption.

The need for careful differentiation between subperitoneal streptococcal infections and appendicitis has been noticed by Connell[2], who quotes J. B. Murphy, "the greatest stress should be laid on the differential diagnosis between intra- and subperitoneal infections".

Acute osteomyelitis of the spine is mistaken sometimes for acute intraperitoneal disease.

Fraser[3], reporting a case of that disorder, notes the resemblance. One of two of my patients suffering with acute osteomyelitis of the spine at first seemed to be suffering from acute peritonitis, and an unnecessary laparotomy was performed. A boggy œdema, which extended into the pelvic mesocolon, was found over the sacrum behind the posterior parietal peritoneum. A correct diagnosis was reached the next day, but in spite of several operations to procure drainage the patient died a month later. At the post-mortem examination it was found that the disease had begun in the body of the 2nd sacral vertebra. In cases of acute osteomyelitis of the spine and in subperitoneal infections the outpouring of inflammatory serum into the extraperitoneal tissues is the cause of the symptoms and signs which mimic those of acute intraperitoneal disease.

An effusion of blood may act in a like manner: A bootmaker, age 48, was sitting in his chair reading a paper. He was suddenly seized with an acute pain in the left iliac fossa, and in the four hours' interval between the onset of his illness and his admission to hospital he vomited four times. He had been invalided from France during the war for 'lumbago and sciatica', and had been treated for this condition by the Ministry of Pensions for some months, but previous to being taken ill was in his usual health. Some years before he had had an operation for a left inguinal hernia.

The patient, a well-nourished man, had a muddy complexion and a furred tongue. A squint obscured his expression, but he was obviously suffering pain. His temperature was 97° and his pulse-rate 76. No abnormal physical signs were found in his chest. His abdomen was distended and its respiratory movements were restricted. The scar of the hernia operation was visible in his left loin, but the hernia had recurred, and a distended sac was seen in the left side of his scrotum.

The abdomen, particularly over the left iliac fossa, was tender on palpation. The hernia contained intestine which was easily reduced, but the patient complained of pain when reduction was effected, and the hernia quickly recurred. The superficial reflexes were absent. The left side of the abdomen was rigid. On percussion the liver dullness extended one finger-breadth below the sixth rib in the nipple line. The right side of the abdomen was tympanitic; lateral to a vertical line situated two inches to the left of the umbilicus, the left side of the abdomen was dull. When the patient was rolled on to his right side the dullness did not shift, and between the tympanitic note on the right side and the dullness on the left the line of demarcation was abrupt and well defined. By rectal examination nothing abnormal was found, and a turpentine enema was returned without fæces or flatus. His urine contained a trace of albumin, but was otherwise normal.

Acute intestinal obstruction was diagnosed, and I thought the cause might be a band in connection with the left inguinal hernia. The abdomen was opened below the umbilicus by a left paramedian incision. The explanation of the physical signs elicited by percussion was then apparent: the descending colon had been pushed forwards and towards the middle line by a large hæmatoma which had stripped up the peritoneum on the outer side of the gut. The descending colon was collapsed, whereas all the intestine proximal to it was distended. The hæmatoma extended from the region of the left kidney to the root of the pelvic mesocolon. The kidney itself, obscured by the large mass of the hæmatoma, was not felt.

The further steps of the operation were improvised on the assumption that the probable diagnosis was a ruptured aneurysm of the left renal artery. An incision was made through the posterior parietal peritoneum over the aorta and a dissection carried upwards; on reaching the left renal vein it was divided between ligatures just distal to the entrance of the spermatic vein. Next, the left renal artery was divided close to its origin from the aorta. The opening in the posterior parietal peritoneum was then closed. The peritoneum on the outer side of the descending colon was now incised, and after clamping and cutting through the ureter, a large mass, consisting of perirenal fat, blood-clot, and the left kidney with its divided vessels, was brought forward and removed. After clearing away the remaining blood-clot, tying all bleeding vessels, and suturing the incision in the peritoneum on the outer side of the colon, the abdomen was closed. The patient recovered. The specimen was examined by Dr. Keith, who reported that the growth was an angioma.

In the foregoing case, a rapid extravasation of blood into the retroperitoneal tissues of the loin produced acute intestinal obstruction. The cause of the obstruction was not recognized to be extraperitoneal, and was sought

for within the peritoneal cavity. Other surgeons have experienced this difficulty in exact diagnosis. Sohn[4] has recently reviewed the history of cases of spontaneous perirenal hæmorrhage reported in surgical papers since 1910, and adds two cases of his own: in one he performed an exploratory laparotomy on the diagnosis of acute intestinal obstruction. Bolland[5], describing a case of spontaneous hæmatoma in sarcoma of kidney, writes: "At 10.0 a.m., August 23, 1922, while walking, she was seized with a sudden violent pain in the left loin, accompanied by extreme weakness, faintness, and nausea. She was taken home and seen by her physician within two hours of the onset of the condition. He found her in a state of collapse, with a very weak pulse, a distended abdomen, and an easily discernible mass in the left loin. She was then vomiting, and on account of this symptom, and the distention, his first thought was of intestinal obstruction." Wood[6] has recorded a case of spontaneous rupture of a hydronephrosis into the retroperitoneal tissues of the left loin, in which the abdomen was opened because "it was thought that the condition might possibly be a ruptured ovarian cyst".

Even should the diagnosis of a subperitoneal effusion be certain, so close may be the simulation of acute intraperitoneal disease that the surgeon may think some intraperitoneal complication has ensued. This is well illustrated by a case of "Rupture of Renal Artery and Vein by Slight Injury" recorded by Miles Atkinson,[7] from which the following extracts are quoted:—

> A boy of 18 was admitted to the Prince of Wales Hospital with hæmaturia. Two days previously he fell while playing cricket and pushed his elbow into his left side. . . . On admission he did not look ill. . . . The whole of the left side of the abdomen and left flank were rigid and tender sufficiently to make examination difficult; but palpation of the left kidney region revealed indefinite swelling below the costal margin which was dull to percussion. . . . His bowels not having been opened since the accident, he was given an enema, with no result. The following morning there was no result from an enema. In the afternoon he suddenly vomited three times, looked rather distressed . . . the abdomen was somewhat distended, tympanitic on the right side, with no evidence of free fluid. . . . The temperature was 101°, the pulse 126. . . . Having regard to the onset of abdominal signs it was thought possible that at the time of the accident there had been a tear of the peritoneum, that since then there had been a gradual leak of urine into the peritoneal cavity, and that now a spreading peritonitis was beginning. . . .
>
> The abdomen was opened by a left paramedian incision. The descending colon was found pushed forward by a large retroperitoneal hæmatoma extending from the region of the left kidney down to the brim of the pelvis. This piece of intestine was small and contracted, but proximal to it the whole intestinal tract was definitely distended. There was no peritonitis. Apparently the large hæmatoma had so interfered with the action of the descending colon as to paralyse it temporarily and cause an intestinal obstruction which enemata had failed to relieve.

Under the title, "Rupture of the Abdominal Aorta: Death from Acute Intestinal Obstruction", Nicory[8] has described a somewhat similar case, in which the hæmorrhage took place into the retroperitoneal tissues on the right side from a hole in the right wall of the aorta 15 mm. above its bifurcation.

In these cases no exact account is recorded of the physical signs which might have been elicited by percussion; but, judging from the records of the operations and post-mortem examinations, there can be little doubt that, had they been looked for, those signs would have been found which were described in the case of the bootmaker, namely, fixed dullness in the loin,

a tympanitic note over the rest of the abdomen, and a sharp line of demarcation between these areas.

Subperitoneal effusions, then, may give rise to symptoms resembling those of acute intraperitoneal disease: large effusions situated in the loin may produce acute intestinal obstruction, but they give rise to characteristic physical signs on percussion. Appreciating these principles, I reached a correct diagnosis in the following cases:—

A post-office employee fell forty feet from a telegraph post on to his buttocks. His pelvis was fractured, and his distended bladder was ruptured, with extravasation of urine into the retroperitoneal tissues of the left iliac fossa. A distended abdomen, with motionless, tender, and rigid walls, suggested intraperitoneal injury. The recognition of the principle that intraperitoneal diseases may be simulated by extraperitoneal effusions, and the detection by percussion of an area of fixed dullness in the left iliac fossa, abruptly separated from a tympanitic note over the rest of the abdomen, led me to the diagnosis of extraperitoneal rupture of the bladder without intraperitoneal injury. The diagnosis was confirmed at operation by finding an extraperitoneal rupture of the bladder on its left lateral wall, and by the absence of any signs of intraperitoneal injury three weeks later at the post-mortem examination.

One afternoon I was asked by my colleague, Dr. Rowland, to see a man of 49 who five hours earlier had been seized with violent abdominal pain. For six weeks the patient had been under treatment for 'left sciatic neuritis'. At first the sciatica was so severe that the patient was confined to bed, but for some days he had been better: arrangements having been made for him to have a course of baths, he was to have travelled to an inland spa on the day he was taken acutely ill. Intending to leave about midday he had a late breakfast and afterwards visited the lavatory. Just after going to stool he was seized with a violent pain in the abdomen and collapsed on the lavatory floor, where, after a few minutes, he was discovered by his wife, who described his appearance as 'blue and covered with sweat'. He was carried back to his bed, and during the next few hours vomited several times.

When I examined the patient he was lying recumbent with his knees flexed; his face was pale and drawn, and he was obviously in much pain and dreaded the slightest movement. He complained bitterly of being unable to pass wind, and said that if he could do this he would be easier. His temperature was 97° and his pulse 120. The abdomen was distended, and barely moved with respiration.

Everywhere to the left of the middle line the abdominal wall was intensely rigid and exquisitely tender, but the most remarkable of the physical signs was the contrast on percussion between a fixed dullness in the left loin and a tympanitic note over the rest of the abdomen. A sharp vertical line of demarcation which could be traced from the costal margin to the pubis, two inches to the left of the middle line, separated the two areas of altered resonance. No swelling was visible, and deep palpation was impossible because of the tenderness and rigidity. The right thigh could be extended with ease, but passive extension of the left thigh was painful. Compared with the right, the volume of the pulse in the left femoral artery was distinctly smaller. On

careful auscultation over the heart, the aorta, and the iliac and femoral vessels, no abnormal sounds were heard. The rectum was empty and collapsed, and no swelling could be felt or any particular tenderness elicited. The urine had a specific gravity of 1020 and contained no albumin, sugar, blood, or pus. The cremasteric reflexes and knee-jerks were present and the plantar reflexes were flexor. A diagnosis was made of a sudden retroperitoneal effusion into the left loin, and the probable source of the effusion was thought to be a ruptured aneurysm. Operative exploration was advised. The patient was admitted to the Royal Berkshire Hospital under the care of my colleague, Mr. Baxter, who, knowing my interest in the case, kindly allowed me to operate while he, in case of need, prepared to carry out a blood transfusion.

The abdomen was opened by a left paramedian incision and the diagnosis of a retroperitoneal hæmorrhage was confirmed. The descending colon was collapsed and pushed inwards and forwards by a large hæmorrhage which had stripped up the peritoneum on the outer side of the colon. The raised peritoneum was then incised, exposing a large hæmatoma which occupied the space between the kidney above and the pelvis below. The hæmatoma was larger below than it was above, and remembering the pain which extension of the left leg had caused, and the diminution in volume of the left femoral pulse, I thought that the probable lesion was a ruptured aneurysm of one of the iliac vessels. The blood-clot was carefully removed and a dissection carried inwards until the psoas muscle came into view, when it was seen that the sheath of the muscle was greatly distended with blood-clot. At a point on the antero-lateral aspect of the muscle, opposite the level of the anterior superior iliac spine, there was a hole in the sheath about an inch long from which blood issued. The source of the hæmorrhage was then sought for in a different direction; palpation along the course of the aorta through the posterior parietal peritoneum revealed a large tense rounded swelling with expansile pulsation at the level of the cœliac axis. By this time the patient's condition was critical, and as control of the hæmorrhage seemed impossible, no further exploration was made.

The abdomen was closed, and a few hours later the patient died. Unfortunately no post-mortem examination was allowed, but it was evident that an aneurysm of the abdominal aorta, or of one of its branches in the region of the cœliac axis, had ruptured posteriorly into the psoas sheath. The hæmorrhage had dissected down the psoas, and after rupturing the sheath of the muscle, had made its way into the loose retroperitoneal tissues of the left loin.

In connection with our subject the following extract from a recent paper on "Injuries to the Kidney and Ureter" by Hamilton Bailey[9] is significant: "In many cases of severe renal injury abdominal distention is seen, and may give rise to difficulty in precise diagnosis. Theoretically, it might be reasonably conjectured that early and obvious signs of a severely damaged kidney would mask an intraperitoneal complication, such as a ruptured intestine, and if this were so, a ruptured kidney might be explored and dealt with effectively via the lumbar route whilst an (additional) intraperitoneal complication was unavoidably overlooked. Between 1893 and 1923 no such case occurred at the London Hospital, from which the comforting conclusion can be drawn that in a given case of severe abdominal injury, if the patient rallies from the

shock and the signs are definite enough to indicate that the kidney has been severely damaged, subsequent meteorism does not, as a rule, imply a dual lesion, and the case can continue to be treated expectantly as far as the peritoneum and its contents are concerned. De Quervain suggested that abdominal distention, following a renal injury, was due to interference with the blood-supply of that portion of the colon overlying the kidney. The records of the London Hospital support this view."

CONCLUSIONS.

1. A rapid effusion of blood, serum, pus, or urine into the retroperitoneal tissues may simulate acute intraperitoneal disease.

2. A sudden and massive effusion into the retroperitoneal tissues of either loin can cause acute intestinal obstruction.

3. A large retroperitoneal effusion in the region of the loin gives rise to a striking and characteristic sign on percussion. Corresponding to the site of a displaced and collapsed ascending or descending colon, there is an abrupt line of demarcation between a fixed dullness in the loin and a tympanitic note over the rest of the abdomen.

REFERENCES.

[1] SHAW, FLETCHER, *Jour. Obst. and Gynæcol. Brit. Emp.*, 1923, xxx, No. 1, 47.
[2] CONNELL, *Surg. Gynecol. and Obst.*, 1919, June.
[3] FRASER, *Lancet*, 1911, ii, 1543.
[4] SOHN, *Surg. Gynecol. and Obst.*, 1921, Dec. (abst.), 497.
[5] BOLLAND, *Ann. of Surg.*, 1923, March, 311.
[6] WOOD, *Brit. Jour. Surg.*, x, 574.
[7] ATKINSON, MILES, *Brit. Med. Jour.*, 1923, i, 324.
[8] NICORY, *Ibid.*, ii, 413.
[9] BAILEY, HAMILTON, *Brit. Jour. Surg.*, xi, 615.

MATERNAL BIRTH PALSY.*

By C. LAMBRINUDI, London.

Within the last six months I happen to have seen two cases of traumatic maternal birth palsy. Since this condition is extremely rare, I thought it was worth while to record them and give a brief outline of the theories of etiology.

DESCRIPTION OF CASES.

Case 1.—The first case was under the care of Mr. Laming Evans at the Royal National Orthopædic Hospital, and shown by him at the Orthopædic Section of the Royal Society of Medicine in November, 1923. She was a primipara, 30 years of age, who had experienced a normal pregnancy. Labour lasted 14 hours. There was a slight general contraction of the pelvis, and high forceps were applied. The child was born alive, though cyanosed and apnœic, and weighed 6 lb.

Immediately on recovering from the anæsthetic the patient felt a numbness and weakness in the whole of her right leg. On getting up three weeks later, she noticed acute pain along the back of the thigh and outer part of the leg and foot; she also had definite foot-drop. The patient when seen five months after her labour showed a paralysis of the toe extensors, the tibialis anticus, and the peronei, with some weakness of the toe flexors. The electrical responses were those of a partial reaction of degeneration.

Voluntary power began to reappear in the tibialis anticus muscle after five weeks' treatment, and, when seen seven months later, she was progressing well.

Case 2.—The second case, under Mr. Trethowan at Guy's Hospital, was a woman of 25. Three years previously she had lost her first child during a difficult labour in which the child's shoulders became impacted. A second pregnancy was normal, but she was examined one month before full term and was noted to have a flat pelvis. For this reason, she was admitted into Guy's Hospital for her labour, which lasted 17 hours. The fœtal head occupied a left occipito-anterior position, and the child was born alive and uninjured without the aid of forceps. It weighed 7½ lb.

During the second half of the labour she noticed very acute cramp in her right leg below the knee. She felt a great relief afterwards, but a feeling of weakness and numbness persisted. On getting up three weeks later, she noticed a right foot-drop.

When first seen she had definite paresis of the muscles supplied by the external popliteal nerve and a dullness of sensation in its distribution. After two weeks the sensation had recovered, but the muscular weakness remained.

ETIOLOGY OF THE CONDITION.

These are two typical cases of traumatic maternal birth palsy. In both the pelvis was contracted and labour difficult; in both the symptoms were definitely associated with labour, and the signs predominated in the

---

* A paper read before the Annual Meeting of the British Orthopædic Association, 1924.

distribution of the external popliteal nerve. Sensation was impaired only in the second case.

There could be no question of the involvement of nerves in an inflammatory exudate, because the symptoms immediately followed the labour, the puerperium was normal, and the vaginal examination was negative. Neither could the foot-drop be attributed to pressure of the external popliteal nerve against the head of the fibula by leg crutches, because, in the first case, the flexor longus digitorum was involved, a muscle which is supplied through the internal popliteal nerve; in the second case no leg crutches were used.

Churchill, writing in the *Dublin Journal of Medical Science* in 1845, quoted two similar cases described by Campbell and Ramsbottom, which were attributed to pressure exercised on the muscles and pelvic nerves during the passage of the fœtal head. He, however, dismisses them with the remarks: "If we recollect the number of severe and prolonged instrumental deliveries which take place without any such result, I think we must reject this peculiarity of labour as a necessary or frequent cause."

The two most quoted papers on the subject are those of Bianchi and Lefèvre, in 1867 and 1876 respectively. They advanced the theory that the paralysis was due to pressure, during the course of difficult labour, of the fœtal head or forceps on the lumbo-sacral cord at the pelvic brim before it enters the plexus. From that time onwards this theory has been generally accepted.

Thomas, writing in the *Johns Hopkins Hospital Bulletin* in 1900, elaborated this theory, and explained the frequency with which the external popliteal nerve is picked out, as follows: "The upper roots of the sacral plexus do not lie on the pyriformis muscle, but against the bony wall of the pelvis, and are thus exposed to pressure during certain difficult labours. It is the dorsal offsets of these roots which lie against the bone and receive the chief injury. The external popliteal nerve is made up of these dorsal offsets and therefore the paralysis is chiefly localized in the distribution of this nerve."

There are, however, certain criticisms to make against this pressure theory.

1. It is difficult to understand how the fœtal head can press on the lumbosacral cord. This nerve lies tucked away in the angle between the sacral promontory and sacral wing, and when there is a discrepancy between the fœtal head and the maternal pelvis, almost all the pressure must come on the sacral promontory and the pubic bones.

2. Were pressure the mechanism, one would expect the paralysis to occur on the side where the fœtal occiput lay. In *Case* 2 the fœtal position was *left* occipito-anterior, but the paralysis was on the *right* side.

3. In a certain number of cases, towards the latter part of pregnancy, the patients complain of definite pains in the distribution of the sciatic nerve. These are not so common as cramps, but, nevertheless, do occur.

I believe that any mechanism which explains this rare post-partum paralysis should also explain the commoner ante-partum sciatic pains. Pressure clearly cannot be responsible for the latter, for if the head has already sunk into the pelvis there can be no pressure, and if not, there can be no pressure without active and painful contractions of the uterus.

In this connection there is an extremely interesting case published by Patel in the *Lyons Médical* of 1905 :—

Case 3.—A woman, age 25, had a normal first pregnancy and labour, but a certain amount of pelvis flattening was noted. The child weighed 5½ lb. She nursed her child for fourteen months, at the end of which she was found again to be five months pregnant. At this time she suddenly felt violent pains in the whole of her right lower limb, and in a few days lost all power in it. After a week the pain subsided, but the paralysis persisted. Within three weeks from the onset the muscles recovered, except those supplied by the external popliteal nerve, which showed the reaction of degeneration. She was, however, able to get about.

The child was born at full term ; low forceps were applied after a 30 hours' labour. The child weighed 5¾ lb. The patient's leg felt lighter afterwards, but the foot-drop persisted. There were no sensory changes. Four months after the birth of the child she was again examined, and the condition was unchanged.

Patel considered this case to be one of mechanical neuritis, rather than of the infective or toxic variety, because there was no evidence of infection and there had been no vomiting or albuminuria. On the contrary, in spite of her disability the patient was able to get about, and, moreover, went to full term. It is difficult to believe, however, that the head of a five months' fœtus could cause such great pressure on the lumbosacral cord.

Within the last month, Brooks read a paper on the sacro-iliac joint before the Guy's Hospital Anatomical Society. He had dissected about twenty pelves of patients who had died shortly after labour, though none of them was difficult. He concluded that during the first stages of labour there was a rotation backwards of the sacrum, the axis being through the lower half of the auricular facet, the excursion in some of these cases being as much as a quarter of an inch. Since this is a mechanism designed to enlarge the antero-posterior diameter of the pelvic inlet, it is not unreasonable to assume that in difficult labour the displacement would be even greater. On the other hand, the lumbosacral cord is known to be one of the tautest nerves in the body.

I would like to suggest, therefore, that the true mechanism of this condition is that of *traction* on the lumbosacral cord, and that the lesion is probably located in the roots of this nerve rather than along its course. This theory would account for both the ante-partum sciatic pains and the rare post-partum paralysis, for the loosening of the sacro-iliac joints, which permits the sacrum to rotate, occurs in the latter part of pregnancy.

Unfortunately, no post-partum demonstration of this lesion in the human being has been recorded, but certain observations in animals are interesting.

Matthews Duncan, in his work, *Researches into Obstetrics* (1868), described the changes that occur in the sacro-iliac joints of the cow. He points out that in this animal the pubic bones are consolidated, and therefore all the movements designed to enlarge the pelvic inlet can only occur in the sacro-iliac joints. He says: "The sacro-iliac ligaments increase not only in thickness but in length, and are thus made slack and yielding. . . . Further, the opposing bone surfaces of the joints are smooth and lubricated. The ilia thus become extensively movable on the sacrum (or vice versa) in an antero-posterior direction, the motion being analogous to flexion and extension in the limbs."

If there is an element of truth about the traction theory which I have suggested, one would expect the cow to be particularly liable to paralytic symptoms before or after parturition, because the movements at the sacro-iliac joints are so very marked and the relations of the lumbosacral cord are not dissimilar to those in man.

Fleming, in his work, *Veterinary Obstetrics* (1896), discusses ante-partum paralysis and says: "Paralysis of the posterior extremities is not rare in the cow, especially when near parturition. It generally occurs 6 to 10 or 20 days before labour. . . . Ordinarily it persists until parturition and then disappears.

"The cause is somewhat obscure, but is supposed to be due to compression or straining of the nerves and vessels of the posterior extremity by the heavy uterus. In five or six cases, there was found at post-mortem examination infiltration of the gluteal muscles and discoloration of their fibres. The spinal canal contained a large quantity of serum, and the membranes of the spinal cord were injected."

In discussing post-partum paralysis, he says that it is comparatively rare. It may follow parturition, especially after the birth of a large calf. "Some authorities think it is due to injury to the sacral and other nerves during difficult labour, the sciatic nerves being particularly liable to injury. Post-mortem examinations, however, only furnish negative evidence of this.

"On the other hand, in some cases the spinal cord has been found injured and its vessels congested, with blood-clots in the spinal canal."

These post-mortem reports appear to favour the view of traction on the roots of the lumbosacral cord, for how else could blood-clot and serum be accounted for in the spinal canal, in a peripheral nerve lesion which is so reasonably presumed to be traumatic? Unfortunately I have not yet been able to confirm them, but should opportunity arise, a lumbar puncture in a recent case would help to settle the point.

In conclusion, I should like to thank Mr. Laming Evans and Mr. Trethowan for their kind permission to quote their cases.

THE ANATOMICAL RELATIONS OF THE GASSERIAN (SEMILUNAR) GANGLION.

By R. D. LOCKHART, Aberdeen.

Anatomy text-books do not place the surgeon in full possession of the details essential to operative procedure upon this region, and in recent volumes of surgery, while the authors write from actual manipulative experience, the anatomical description and illustration are inadequate for those who have not previously attempted to "beard the ganglion in its den!"

The four-poster pituitary bed is canopied and securely side-draped by dura mater, and the pituitary body itself is slung in a hammock suspended from the periphery of the canopy, as can be seen in the coronal section through the sella (*Fig.* 325). Between the wall of the hammock and the side curtain is the cavernous sinus.

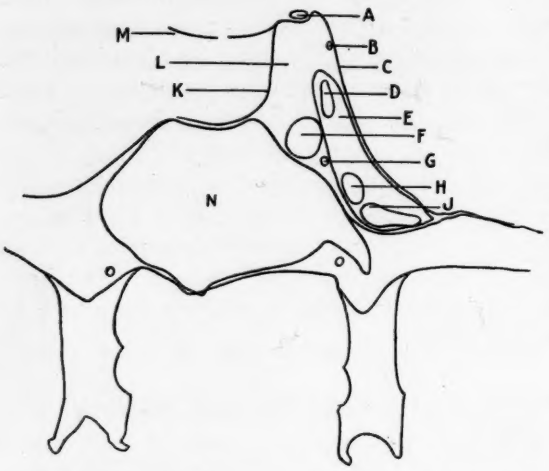


Fig. 325.—A, 3rd nerve; B, 4th nerve; C, Side curtain; D, First division of 5th nerve; E, Meckel's cave; F, Internal carotid artery; G, 6th nerve; H, Second division of 5th nerve; J, Third division of 5th nerve; K, Hammock; L, Cavernous sinus; M, Canopy; N, Sphenoidal sinus.

The tentorium cerebelli splits at the postero-superior border of the petrous bone, the upper layer becoming the dura mater of the midcranial fossa and also the side curtain, while the lower one covers the posterior aspect of the petrous bone. Where these layers separate along this border of the bone, the superior petrosal sinus runs to spread out like an estuary into the cavernous sinus, but just at the estuary the dura of the posterior aspect of the petrous bone is invaginated above the apex of the bone beneath the superior petrosal sinus and beneath and medial to the side curtain. This invagination of dura mater goes by the name of Meckel's cave. Narrow at its entrance, where it transmits the roots of the fifth nerve, the cave widens to accommodate the ganglion with its branches; and a probe inserted along the lateral wall of the cave reaches forwards to the anterior wall of the pituitary fossa, laterally to the foramen ovale, and upwards, fully half the height of the cavernous sinus, without much resistance. While the lateral side of Meckel's pocket is easily freed from the ganglion and its branches, the medial wall is adherent, particularly to the first division and the anterior

part of the ganglion. Again, the lateral wall is fused with the side curtain, but these layers may be dissected apart (*Fig.* 326), and it must be remembered that they are separated posteriorly by the superior petrosal sinus.

Bearing in mind that fully the lower half of the lateral wall of the cavernous sinus is formed by three layers of dura mater, of which the outer two are fused, it is easily possible, by an intradural approach, to incise these two layers for exposure of the roots, ganglion, and branches, and this without tapping any venous sinus, provided the incision is not carried as far back as to enter the superior petrosal sinus, which actually separates the two layers near the neck of the cave.

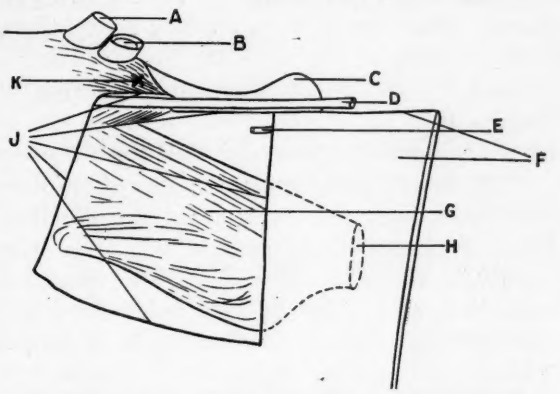


FIG. 326.—A, Optic nerve; B, Internal carotid artery; C, Dorsum sellæ; D, 3rd nerve; E, 4th nerve cut away in flap; F, Free margin and upper surface of tentorium cerebelli; G, Meckel's cave exposed from lateral aspect by removing flap; H, Entrance to cave; J, Cut edges of flap; K, Anterior clinoid process.

In certain cases (*Fig.* 327) the cave reaches as high as and fuses with the canopy, and the sphenoparietal sinus tunnels back between the side curtain and the cave, to enter the cavernous sinus together with the superior petrosal sinus.

The present preference to cut the sensory root may render recognition of the motor root important in bilateral cases of trigeminal neuralgia. In addition to its identification by stimulation, it may be defined by its position superior and medial to the sensory root in the neck of the cave, and then running diagonally underneath the ganglion to the foramen ovale, although observers have seen it transmitted separately through a small foramen medial to that in question.

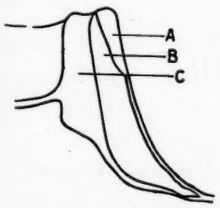


FIG. 327.—A, Sphenoparietal sinus; B, Meckel's cave; C, Cavernous sinus.

It has been stated[1] that the foramen spinosum with its middle meningeal artery frequently lies on a plane anterior to that of the foramen ovale, but the examination of 350 specimens has failed to find this variation.

When the floor of Meckel's cave is removed the bone is not bared but covered by the periosteal layer of the dura mater through which a branch of the middle meningeal artery supplies the ganglion, and the great superficial petrosal nerve runs beneath the ganglion to its pterygoid (Vidian) canal.

It will be noted that the third nerve has been purposely excluded from the cavernous sinus in the coronal section through the pituitary fossa (*Fig.* 325). Most text-books illustrate the nerve within the sinus in this area; but it begins to tunnel the dura mater at the level of the anterior clinoid process, and, in any case, does not enter the sinus until after it has dipped under this process, otherwise Horsley would not have injured

this structure in his operations upon the pituitary body by the temporal route.

Fig. 325, showing the structures resting upon the doorstep of the pituitary fossa, suggests the idea that, by burrowing under the dura mater of the mid-cranial fossa, the surgeon might break through this doorstep into the sphenoidal sinus, and gain the sellar floor from below in suitable pituitary cases. This would be a shorter route than any other. The bone is usually transparently thin; indeed, the doorstep and sellar floor have been seen formed by the sphenoidal sinus mucous membrane alone. Such a case as the one illustrated would lend itself to the production of trigeminal anæsthesia by painting the sphenoidal wall with cocaine.[2]

It is an interesting etiological consideration whether sphenoidal sinus conditions may not be associated with the lower position of the second and third divisions of the fifth nerve upon the doorstep and the occurrence of neuralgia in these very branches; and further, interesting to associate the fact that the sides of the sphenoidal sinus are seldom symmetrical with the fact that bilateral neuralgia is of infrequent occurrence.

REFERENCES.

[1] FRAZIER, C. H., *Keene's Surgery*, v, 964.
[2] *Proc. Roy. Soc. Med.* (Laryngol. Sect.), 1924, March 7.

FACTS AND CONSIDERATIONS IN A STUDY OF THE THYROGLOSSAL TRACT.

By A. P. BERTWISTLE, Leeds.

WITH AN ACCOUNT OF THE EMBRYOLOGICAL CONDITIONS.

By J. E. FRAZER, London.

GROWTH AND DEVELOPMENT OF THE THYROGLOSSAL TRACT.

In giving an account of the changes undergone by this outgrowth it will not be necessary to enter into the details of all the formations connected with it. It is a matter of common knowledge that there is such a growth, that it arises immediately behind the tuberculum impar, and that it is concerned ultimately in the formation of the isthmus and lateral lobes of the thyroid gland; but there are certain features and particulars in its development that are not so generally known or appreciated, and it is with these that I wish to deal in the present communication. It may be added, moreover, that, the median growth being the subject of this paper, the writer is only incidentally concerned with the formation of the lateral portions, and does not propose to enter on any further description of their development.

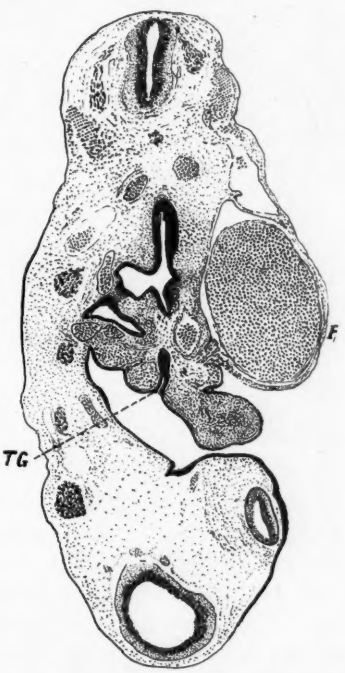


Fig. 328.—Section through the head of an embryo of 4·5 mm. TG, Thyroglossal outgrowth; P, Pericardium and left auricle, cut.

The median thyroid bud is a very early formation, becoming evident as soon as the mandibular arch is definitely present, and retaining its association with the entodermal lining of the primitive pharynx usually up to the time when the embryo measures some 7 or 8 mm. in length; but there appears to be some little variability in this matter. The section shown in *Fig.* 328 illustrates this continuity in an embryo of 4·5 mm., and shows also the shortness of the outgrowth at this stage and the presence of a definite lumen in its proximal part. The section is very oblique, owing to the lateral flexion of the head of the specimen, but the small second visceral arch can be recognized behind the outgrowth, while the hollow appearance in front of it, between it and the mandibular projection, is produced by the line of section here passing through the groove beside the tuberculum impar.

The obliquity also brings other things into evidence, however, and it is really to show these that the section has been taken. It can be seen that the pericardium (P) has been cut on one side, with the left auricle, and the aortic stem coming from this is cut across close to its division into right and left ventral aortæ. The thyroid bud is in relation with these, lying against the arterial trunk and just above the pericardium; this last relation would be more evident in a sagittal section. The relation of the outgrowth to the arterial trunk and pericardium is an early and important one, concerned in its subsequent displacement and secondary position. The bud, as soon as it grows in from the entoderm, comes against the place of division of the arterial trunk (which is at the time immediately below the site of its formation), and retains this relationship as long as it is free.

Fig. 329 shows a sagittal section through an embryo of 10 mm. The median outgrowth is seen in contact with the arterial stem and also with the upper and front part of the pericardial wall, but its continuity with the entoderm is broken, although it has clearly not moved very far away from its place of origin. An important fact now presents itself. The bud and the arterial stem are lying in a mesodermal bed decidedly 'looser' in texture than the condensed masses which intervene between them and the pharyngeal cavity and form the visceral arches. This is seen in the section, and is also very apparent in the section shown in *Fig.* 328, and the fact is one of primary importance as rendering possible the dislocation (with reference to the visceral arches) in a caudal direction of the structures lying in the looser lower bed of mesoderm. It is evident that an epithelial bud lying between condensed masses, such as the visceral arches, would remain in that situation, as it possesses no power of active movement other than that extension in area brought about by rapid multiplication of its cells; hence such a bud must pass beyond the constraining influence of the condensed masses, and enter an area where it is not subjected to so much pressure, and where, if its further development calls for change of position, this can come about through the action of some efficient agent. This is what has occurred in the case of the thyroid bud; growing in the middle line, with the mandibular condensation in front and the developing 2nd arch forming behind, it can only extend down between these condensations, and thus very soon reaches the plane of less condensed mesoderm which underlies the arch-masses, coming into relation here with the common arterial trunk where it divides.

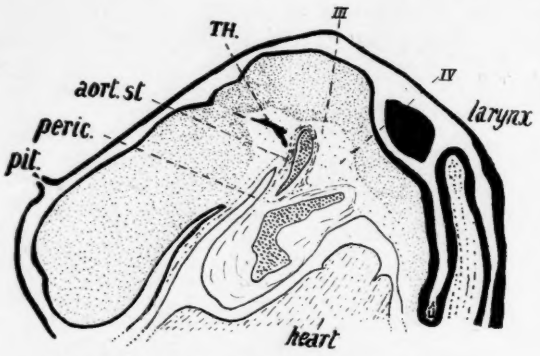


FIG. 329.—Median sagittal section, 10-mm. embryo. Reconstruction. TH, Thyroglossal growth; *peric.*, Pericardial cavity; *aort. st.*, Aortic stem; *pit.*, Rathke's pouch; *III, IV*, general lines and positions of 3rd and 4th lateral pouches.

The plane of looser mesoderm is no doubt a remnant of the original floor of the foregut. The new floor is made by the visceral arches, forming

immediately below the entoderm of the cavity, invading, in a sense, the floor-area from the sides. The old mesodermal layer remains associated with the roof of the pericardium, and the common arterial stem enters it from the pericardium and divides within it. The arterial arches enter the condensations of their corresponding visceral arches, and are thus retained in position. The lateral pouches of the pharynx, projecting ventrally and laterally between the arch condensations, also come to lie on each side within this bed of looser mesoderm, ventrolateral to the pharyngeal cavity. It is of interest to note that the 3rd and 4th pouches, owing to the curve of the pharynx, are near one another, and not very far from the arterial stem, although, of course, they are placed more laterally; thus, in the 10-mm. embryo (*Fig.* 329) the general positions of these pouches might be indicated by the interrupted lines *III* and *IV*, and their nearness to the arterial stem, the thyroid rudiment, and the pericardium is evident.

Growth in length—and in other ways—of the pharynx is now proceeding apace. It is usually said that the pericardium and heart assume a more caudal position with reference to the pharynx, it being implied that this retrogression is primarily a shifting of the heart and its surroundings. This, however, is not the correct way of viewing the matter. The pericardium should be looked on as a fixed structure, being continuous with the septum transversum, which makes its caudal wall; it cannot shift caudally, and the description usually given is incorrect in so far as it implies such shifting. Actually it is the excessive growth of the anterior end of the embryo which carries it beyond the stationary pericardium, curving round this at first as it is attached to its ventral surface, but gradually standing away from it as the attachments lengthen. So far as the neck is concerned, this growth forwards of the pharyngeal floor is rendered possible by the existence of the looser stratum between it and the pericardium, the hinder part of the floor moving up on the underlying structures. Thus the thyroid rudiment and the arterial stem retain their general relationship with the pericardium, and the pharyngeal pouches, moving up with the arch structures that make the floor of the pharynx, come into close relationship with them. This is seen in *Fig.* 330, a sagittal section in an embryo of 13·5 mm. The relative positions of the arterial stem, the thyroid rudiment, and the pericardium are practically as before, but the whole pharyngeal floor, greatly thickened, has moved up over them, and the thyroid growth has extended laterally and has come into relation with the 4th pouch, the general position of which at this stage is indicated by the interrupted line *IV*.

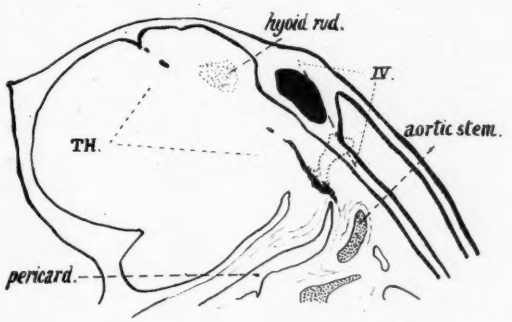


FIG. 330.—Median sagittal section, 13·5-mm. embryo. Reconstruction. TH, Remnants of thyroglossal tract, the line of which is ventral to the hyoid rudiment. *IV*, General line of the 4th lateral pouch.

This, then, is the way in which the change in level comes about; but

before passing to the consideration of the extension of the outgrowth it will be convenient to deal with one or two other matters which are evident on examination of the sections already shown. It is clear, as can be seen in *Fig.* 329, that the outgrowth reaches the deeper loose layer by passing between the condensations of the 1st and 2nd arches, and its subsequent position is on the ventral side of the 2nd arch; as the pharynx grows, the 2nd and succeeding arches are moved forward over the thyroid rudiment. The pharyngeal growth has already progressed to a slight extent in the 10-mm. specimen, and the thyroid rudiment, apparently as a consequence, is separated from the entoderm; this matter will be referred to later. The skeletal bars of the 2nd and 3rd arches, the constituent parts of the hyoid bone, are formed in the mesodermal condensations of these arches. They are not in any way evident before, or for a considerable time after, the rudiment has separated from the entoderm, but, although on this account the direct demonstration cannot be given, it seems to me to be quite clear that the track of the growth is, and must be from the beginning, *ventral* to the hyoid structures. At 12 mm. the condensations of the skeletal bars are evident, and their situation in the middle line (3rd arch) is indicated in the 13·5-mm. specimen in *Fig.* 330. The relation to the arterial stem and pericardium is not one of absolute contact, suggesting that the relation is maintained, after its attainment, because there is no force acting on the rudiment in such a way as to displace it.

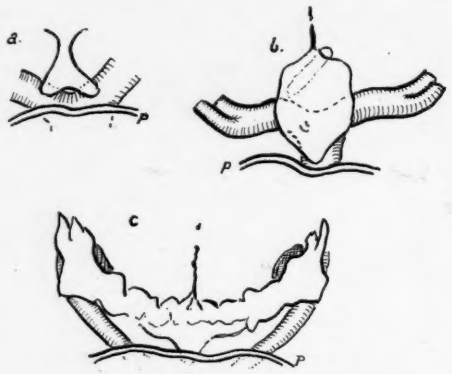


FIG. 331.—Thyroid growths seen from the front. Reconstructions. Embryos of (*a*) 4·5 mm., (*b*) 10 mm., and (*c*) 13·5 mm. The relation of the growth to the arterial trunk is shown, and to the roof of the pericardium, P.

Turning now to the growth itself, its appearance from the front in each of the three stages chosen is given in *Fig.* 331. The bilobed state of the first stage becomes changed into the nearly symmetrical broad shield of the second, which ends below in a blunt median point near the pericardium. The shield is extending on each side along the arterial trunks. In the 13·5-mm. specimen the extension along the arteries has brought the growth into relation with the 4th pouch, to which it seems to be definitely attached. This attachment, then, may be taken as an agent anchoring the growth on each side. In the middle it may be presumed that the original relationship with the central vessel and pericardium persisted until the attached lateral portions interfered with it; hence we find the central portion lower than the rest, still with a central stem running into its upper part and a blunt point below, near the pericardium, but the face of the original shield is changed by the proliferations it has undergone.

Fig. 332 is a schematized reconstruction showing the whole extent of the thyroglossal tract in an embryo of 18 mm.; in this, for the first time, the upper stem of the 'shield' is found lying on one side. The curve of the extended growth is much closer than before, the arterial division is further

away, as is also the pericardium, and there is no definite indication of the blunt-pointed lower end.

Proceeding now to the consideration of some of the details brought out in these figures, we may turn first to that part of the tract lying above the hyoid bone. This seems to disappear almost entirely in the earlier stages, after the separation from the entoderm, but I am inclined to think that the cells of this part of the tract, though it is almost certainly broken up, are not really lost. My chief reasons for this belief are: (1) There is no sign of degeneration or destruction in the cells of the tract whenever they can be definitely found, although their staining reactions are very slight or absent; (2) The position of the tract can be followed without very much trouble, and groups of cells can be found in it here and there, which may be, and in my opinion probably are, remnants of the tract; and (3) That in somewhat later stages darkly-staining cells like the other cells of the tract are found in its line. Such definite remains are seen in *Fig.* 330, and a very complete and undoubted tract in *Fig.* 332. The remnants—that is, the formations that seem to me to be remnants—are few and scattered. They appear to be most often found near the upper end of the track of the outgrowth, but sometimes one sees them just in front of the body of the hyoid. These situations are what might be expected, and I believe that the remnants are usually few and scattered, as there is much individual variation in my specimens of the second and third months. The 18-mm. embryo (*Fig.* 332) is the only one that I possess showing a continuous tract reaching the hyoid, but that such rare tracts are found after birth is shown by such cases as that of C. F. Marshall (*Jour. of Anat. and Physiol.*, 1891).

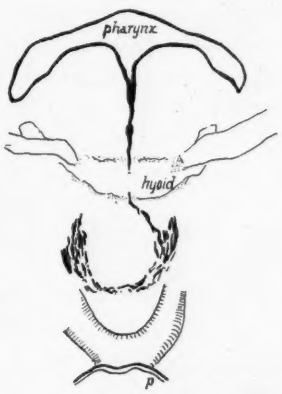


FIG. 332.—Reconstruction of the line of the thyroglossal tract in an embryo of 18 mm., showing an unusually complete suprahyoid portion, and the obliquity of the lower tract, produced by the migration of the proliferating cell-masses of the central (isthmus) region, with which it is continuous, to join the lateral mass of one side.

The tract below the level of the hyoid is present in all my specimens of the second and third month, in the middle line, or, more commonly, to one side or the other; it can usually be picked up just above the lower border of the hyoid bone and followed down from this point. I have also found it in nearly all the fœtuses that I have examined from this point of view, and it is of interest to note that Marshall (*Jour. of Anat. and Physiol.*, 1895), in 60 children aged from a few weeks up to 10 years, found it (pyramidal process) in 43 per cent. This part of the tract consists of the unpaired and originally central stem, and the isthmus and lobes; of these the stem and isthmus are the portions of more immediate interest in this place. These are the remains of the earlier 'shield' after its lateral prolongations along the arteries have reached the lateral pouches. When this stage is reached the stem begins to show some indications of proliferation, and of breaking (*Figs.* 331 and 332), but much more definite signs of proliferation are found in the isthmus. The columns and masses of cells formed in the central isthmus move in great part to the lateral

lobes, and form parts of these, and in this way, the frequent obliquity of the pyramidal lobe, or stem, is probably brought about; the result on the really central stem of such growth is shown in an extreme degree in *Fig.* 332. Looked at in this way the position of the stem in any individual case might be said to be accidental, and we have an explanation at once of the inequalities of the isthmus, its great size in association with a median pyramidal lobe, its absence with a pyramidal process attached to one lateral lobe, the bifurcation of the lower end of the pyramid with attachment to both lateral lobes, and in fact of all the varieties figured by Marshall. The stem may remain as a complete pyramid, or it may be broken; some of the sections show it clearly broken, especially in its upper part. The cells thus left in position are demonstrably capable of the staining reactions exhibited by the continuous parts, and one might be justified in looking on them as potential thyroid fragments which might conceivably (as, for instance, if the rest of the thyroid tract were not functioning adequately) take on further development as part of the thyroid structure. Such groups of cells might well be expected more especially near the hyoid bone, but, in the absence of a pyramidal lobe, might occur anywhere in the line which it had occupied before it broke up and apparently disappeared.

It is not possible to make any decided statements about the blunt-pointed lower end of the shield of earlier stages. It is to be recognized in embryos of 15 and 16 mm., but not in any of the later specimens. There are small veins forming a plexus round this part of the growth, however, and it seems to me not improbable that proliferating cells may extend along these, thus reaching the innominate veins and even the pericardium, but such cells have not been recognized definitely. In the 16-mm. specimen there are some projections downwards from this region on one side, and there is a suggestion of cells coming off these and applying themselves to the thymic rudiment. Thyroid formations do occur in the upper thorax, and the means by which they find their way to this region may be as indicated above, but I am not at present able to produce clear evidence on the matter.

A couple of points still remain for consideration, the first of which has to do with the relation of the tract to the hyoid bone. This relation I hold to be primarily *ventral.* I have already spoken of the relation the tract must necessarily bear to the condensations of the arches if these are to be free to move forward over it, and it is only necessary to understand that the skeletal elements are formed in these condensations to see the necessary relationship. It is unfortunate that, in the human embryo, the tract is broken before the hyoid rudiments are recognizable, but a glance at the reconstructions given will show the relation. Moreover, sometimes the indications are more definite, and I have before me now the photograph of a sagittal section through the region in a 12-mm. embryo in which there does not seem to be any doubt whatever of the ventral position of the tract. I may add that His and, I think, most embryologists of to-day have this opinion on the matter, and that I reached it independently years ago as the result of direct observation. Such cases as Marshall's (1891), in which he described the remnant as passing up to the tongue dorsal to the hyoid are, I am convinced, founded on errors

of observation, and in that case he describes and figures the supposed continuity as broken and incomplete. The very few opportunities one has had of dissecting the condition in the adult have borne out completely the findings in the embryo and fœtus. The retrohyoid part of the tract—which is usually present—has been carried into this situation by folding round the lower edge of the bone;

FIG. 333.—Diagram to show relation of a complete infrahyoid tract (T) to the hyoid bone. Beginning in front of the bone, it bends backwards round its lower border, and turns up behind it for a little distance before passing down. This is a secondary turn, produced during the third month from the earlier and purely ventral condition.

the complete tract would be as represented in the scheme in *Fig.* 333, but it may be broken in various ways.

This disposition seems to come about in the third month, and is associated with the general settling down into place of the structures in the neck as this reaches its final stages. One has seen the tract where it lies near the side of the body of the bone partly enclosed by bony growth round it, but careful dissection showed the true nature of the condition and exhibited a complete folding such as that in the scheme. Such a condition might perhaps lead to the description of a tract passing through the bone, for which otherwise no justification is apparent. The relation of the tract to the cartilaginous hyoid body in the third month is of interest, for it is closely related to it, embedded almost in the perichondrium of its lower and front part; this may account for its occupying a notch on the bone, and, if one may venture on a surgical matter, it may point to the advisability of removing the periosteum in the effort to excise the area of the tract.

The second point has to do with the foramen cæcum. This, although it gives the situation *on the surface* of the spot from which the thyroid growth arose at one time, is not really the remnant of that spot at all. In the third month certain posterolateral growths form the hinder part of the tongue. It is not necessary to enter into an account of these, and it is sufficient to say that, growing on to and over the corresponding aspect of the tongue, they meet in the middle line behind, and extend outwards and forwards on each side from this. The angle of their divergence makes the foramen cæcum, the angle being situated over the site of the thyroid growth. The two parts, meeting just behind this, fuse together, but in doing so they enclose a space deeply between them. A section made horizontally through the tongue in an embryo of 35 mm. is given in *Fig.* 334, showing the meeting of these two portions of the tongue over a cavity, from the floor of which, but at a lower level than that of the section, the epithelial projection that marks the old thyroid tract is to be found; the cavity is evidently a secondary one, superficial to the old surface, and is lined by epithelium of the surface. This cavity appears to dwindle in size quickly; it is a small recess in a specimen about 38 mm., with its lining membrane reduced to a double layer of cells, and it apparently disappears shortly after this. It is not impossible, however, that the cavity, like others made by enclosure, may persist. Certainly a depression some $\frac{1}{4}$ in. or more in depth can be found every now and then

below the foramen cæcum, and it was probably an extensive persistence of this cavity, extending back on each side towards the lateral epiglottic regions, that Heister described in 1750 as a new system of 'salivary ducts'. If the cavity persists, it opens on the surface at the foramen cæcum, and thus the thyroid tract, running from its floor, comes to 'open' indirectly at this foramen. If persisting and closed, on the other hand, it will be superficially separated from the mucous membrane only by a thin layer of submucous tissue.

It has seemed to me quite possible that this may be the origin of some of the so-called thyroglossal cysts placed superficially on the hinder part of

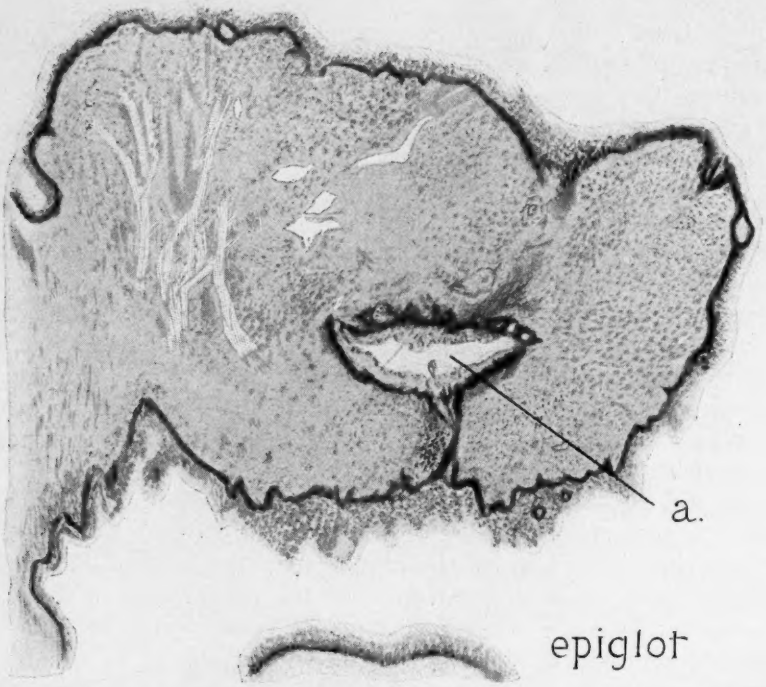


FIG. 334.—Horizontal section through upper and back part of the tongue of an embryo of 35 mm., to show the secondary cavity (*a*) enclosed by the meeting of the posterior tongue-growths in the middle line.

the tongue; if this were proved to be so, they would be vestigial retention cysts with no thyroid or thyroglossal character, unless indeed there were concomitant activity in a thyroglossal stalk-remnant attached to the lower wall of the cyst. *Case* 10 of this series seems possibly a vestigial retention cyst of such a sort.

Summing up from a clinical or pathological point of view the main facts dealt with in this account, we may say that:—

1. The thyroglossal remnants above the hyoid are few, and if present are usually either near the foramen or near the hyoid.

2. Below the hyoid the tract is present completely in many cases, incompletely, i.e., potentially, in others.

3. The complete infrahyoid tract extends from the hyoid, usually folded up behind it from below, to the thyroid gland, either centrally or on one side.

4. The incomplete tract has *potentially* exactly the same line; it is the tract simply broken up, and its parts remain *in situ*.

5. Any of these remnants may take on further development, especially perhaps if a condition of relative hypothyroidism exists.

6. It is possible that some cells of the tract may be separated off and lodged below the level of the gland; if this is so, these cells may also be liable to take on further development. It may be pointed out here that certain continental embryologists, some thirty to forty years ago, maintained that the median fistulæ, etc., found low down in the neck, arose from remnants of the precervical sinus, a view strongly supported by Kanthack in this country. This view would not receive extensive support from embryologists to-day, and may be passed by.

7. Cysts lying superficially below and behind the foramen cæcum may have a possible origin from a space included between the two halves of the developing hinder part of the tongue, and not necessarily be connected at all with the thyroglossal tract.

It may be added, finally, that any appearance of a potential 'duct', judging from the arrangement of cells, seems to be confined to the suprahyoid portion. The lower stem, although it appears to be but a separated part of the 'duct', does not show a tendency to arrange its cells in a similar way. When it enlarges it proliferates like the lower structures, and, when broken, the separated cell groups may be presumed to have the same tendency. Hence it seems to me that such a thing as a dilated duct, or fistula from such a remnant—if it occurs at all—would be above the hyoid, but that in most cases, those which occur below the hyoid, the first formation would be in the shape of a thyroid growth with development of the usual thyroid cysts or vesicles, and any secondary fistula would open into such a cyst. Though the possibility of the other formation cannot be denied, these appear to be the rational inferences from what I have observed of the development, and my small experience of the pathological conditions seems, so far as it goes, to support this view. [J. E. F.]

CLINICAL CONSIDERATIONS.

The unsatisfactory results of operations performed on the cysts and fistulæ of the thyroglossal tract are well known. Some of the factors leading to these results are discussed above, especially the anatomical relations of the thyroglossal tract to the hyoid bone. The increased discharge from the fistulæ which occurs during the ingestion of food raises the question as to whether the thyroid gland and its congeners become more active during the process of digestion. Only eleven cases are described, but as they illustrate most of the points which have been discussed, it is felt that their publication is justified. They have all been personally investigated, with the exception of *Case* 10, the details of which were kindly supplied by Dr. Vining.

FISTULÆ.

Case 1 (*Figs.* 335 and 336).—A woman, age 28, was admitted with a small translucent nodule in the neck, exuding a thin serous fluid. She states this secretion was increased during mastication. According to her account, it had existed about six years, during which time it had occasionally ceased discharging. A swelling then appeared. This burst, and the immediate discharge was a thick, yellow fluid. On puncturing for injection purposes a honey-like fluid escaped.

OPERATION.—A transverse incision was made, and the sinus, which ended above in a fibrous cord, was dissected out from the deep tissues between the hyoid bone and thyroid cartilage, and the wound closed. Healing was delayed, but six months later there had been no return. A slight keloid was present.

FIG. 335.—*Case* 1. Photograph of sinus.

MICROSCOPIC EXAMINATION.—An open tract runs along the length of this specimen, lined by very vascular and thick granulation tissue. No epithelial lining cells are to be seen anywhere. A fairly thick mass of thyroid tissue, with well-developed vesicles, extends along the whole length of the tract, but only along one side of it (? which side). Every stage of formation of the vesicles is to be found in the sections of this aspect of the tract.

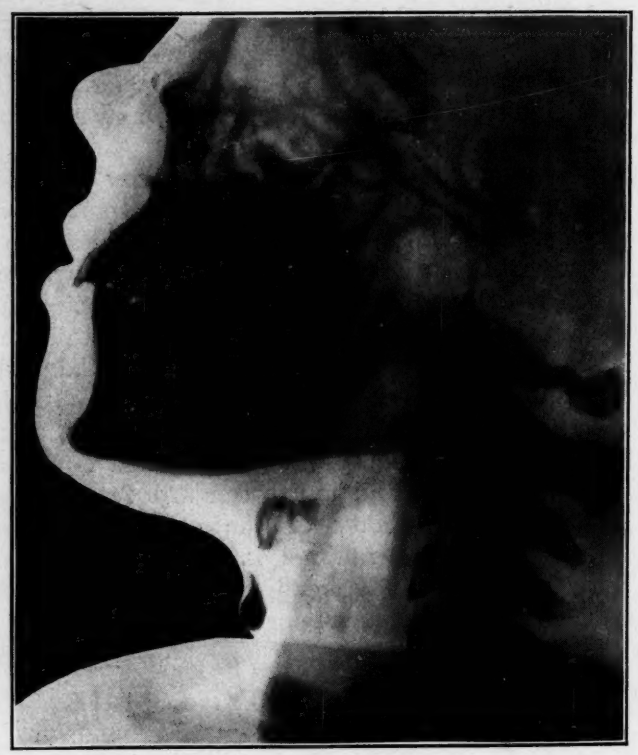


FIG. 336.—*Case* 1. Radiograph showing pear-shaped bead of bismuth paste.

Case 2 (*Fig.* 337).—"Occurred in a boy aged 7. The sinus was first noticed when he was 18 months old, and has since remained stationary. At times a swelling appeared, and then he had pain during mastication, otherwise there was no inconvenience except from a thin discharge, occasionally replaced by a sticky yellow fluid. The sinus was present in the mid-line of the neck, surrounded by cicatrices. It moved on deglutition, and was palpable as a firm cord attached to the hyoid bone.

"'B.I.P.P.' was introduced into the sinus by means of a large bone syringe. The opaque material is seen passing up towards the hyoid bone in the radiogram."[1]

OPERATION.—The sinus was found to end in a fibrous cord which passed into the median raphé above the hyoid bone. Three months later the sinus was still present, but the discharge less copious ; a keloid had developed.

MICROSCOPIC EXAMINATION.—Sections taken throughout the growth show that it consists mainly of thick fibrous tissue, arranged in bundles. Pockets and strata of somewhat embryonic-looking tissue lie between and among these bundles, and in these occur clumps of large and darkly-staining cells, which here and there form small cysts or groups of two or three cysts : in some instances there seems to be colloid in such. The large cells in the pockets seem to have a tendency to arrange themselves circularly, and the appearances are reminiscent of embryonic thyroid formation. No duct or surface is to be found in the specimen.

FIG. 337.—*Case* 2. Radiograph of sinus injected with 'B.I.P.P.' Observe relation to hyoid bone.

Case 3.—This was a girl of 4, and the ulcer first appeared a few months after birth. There was constant discharge, except on the rare occasions when it healed over, only to break down again. Several attempts had been made to deal with it.

OPERATION.—A similar operation was performed to that of *Case* 1. The wound, however, broke down ; four months later the discharge was as copious as ever, and a slight keloid was present.

MICROSCOPIC EXAMINATION.—The specimen is rather fragmentary, but there seems to be a cavity or duct within it, with walls of granulation tissue, but showing in places an epithelial lining. This is of single cells in part, but becomes stratified in other parts, and has the appearance of invading the underlying tissue, having in fact a suggestion of malignancy here and there. The surrounding tissue is composed of bundles of fibrous appearance. No structures that can be recognized as probably thyroid in nature can be found in the intervals between the bundles, where there are small masses of darkly-staining cells of somewhat embryonic sort ; these masses appear to be continuous in places with the epithelial lining of the sinus. A few muscle bundles are present.

CYSTS.

Case 4 (*Fig.* 338).—A woman, age 29. The swelling of the neck which had existed for some time had lately increased in size, and become tender, interfering somewhat with mastication.

OPERATION.—A transverse incision was made along the line of the natural crease in the neck. The cyst was freed as much as possible, not, however, without the contents escaping. The fluid in the cyst resembled pus ; some was collected for chemical examination, and found to contain iodine. The whole of the cyst wall was carefully removed, and the wound closed by intradermal suture. There was no return of the cyst after four months.

MICROSCOPIC EXAMINATION.—One slide. A section showing thick granulation tissue round what may have been a sinus or cavity ; outside this are bands and

bundles of fibrous tissue, with material of embryonic type between the bundles. A tendency to formation of thyroid structures is found here, with some groups of definite and fair-sized vesicles.

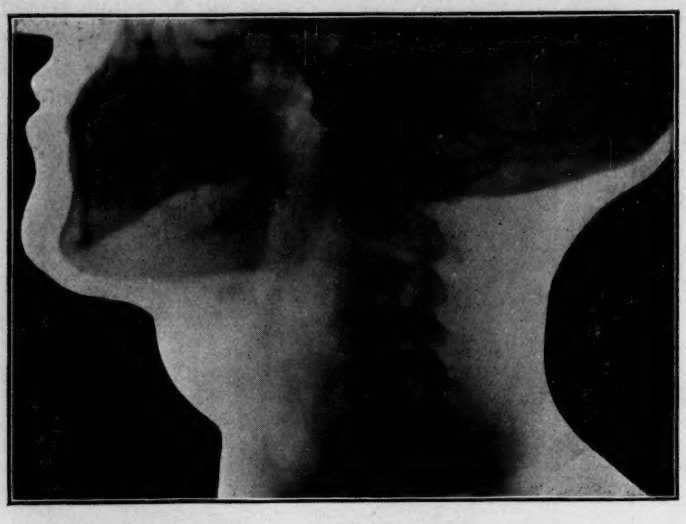


FIG. 338.—*Case* 4. Note prominence in neck due to cyst.

Case 5 (*Figs.* 339 and 340).—A girl, age 12, was admitted on account of a swelling in the neck that somewhat obscured the hyoid bone. It was tender and rapidly increasing in size. The cyst was aspirated preliminary to the injection of collargol. This had for its purpose the demonstration of the cyst's extent radiographically, and the destruction of any bacteria present. The contents were like pus. Unfortunately the swab taken was lost, and no bacteriological report was obtained. Chemical analysis showed the absence of iodine. After the injection she became faint, and operation was delayed until next morning.

OPERATION.—A transverse incision was made along the natural crease of the neck. All the tissues were found to be sooty black in colour. Extreme difficulty was experienced in dissecting the cyst from behind the hyoid bone. A subcuticular suture completed the operation. The following day a black discoloration was apparent in the neck. Later a temperature of 101° developed, and a few days afterwards the wound broke down, gaping one inch. The pigmentation had spread over the adjacent part of the face, and 5 c.c. of antistreptococcal vaccine were injected. A week later it was beginning to fade, but the wound was unhealthy and its surroundings were brawny. At the end of six weeks the wound had almost healed, but the tissues in the vicinity were still indurated. Four months after operation there had been no return, and the scar, although adherent to deep structures, was surprisingly inconspicuous, being situated in the natural crease of the neck. The pigmentation disappeared about the third week.

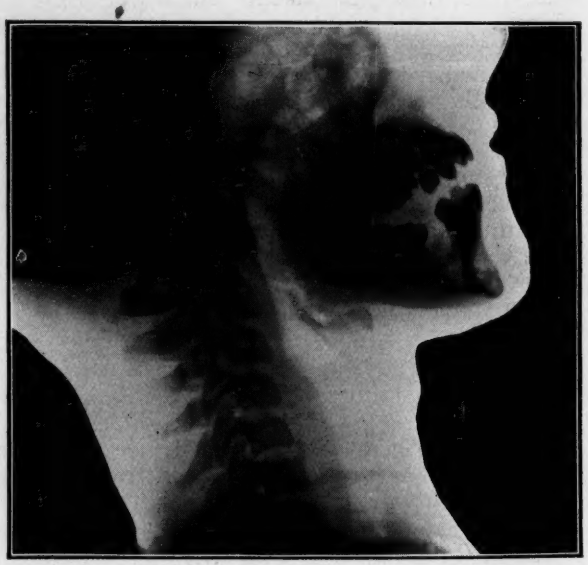


FIG. 339.—*Case* 5. Showing prominence in neck.

MICROSCOPIC EXAMINATION.—Mainly a mass of fibrous tissue, with collections of large cells with darkly-staining nuclei between the fibrous bundles. No tendency

to vesicular formation is to be seen in these cell-groups for the most part, but such is to be found, however, here and there near the walls of a cavity occupying one side of the specimen. This cavity is irregular, with ragged walls of apparently necrotic tissue, surrounded by much infiltrated substance. There is no sign of brown injection in this cavity, but this is to be found in a couple of recesses—whose connection with the larger cavity was not discovered—and the walls of these recesses are composed of fibrous tissue without any signs of small-cell infiltration.

FIG. 340.—*Case* 5. Cyst filled with collargol.

Case 6.—A girl of 14 had a cyst in the middle line of the neck adherent above to the hyoid bone. It was causing some disfigurement, and had recently increased in size.

OPERATION.—A vertical incision was made, and the cyst removed. It was adherent below to the thyroid gland. One month later there was a recurrence. A long red scar was visible, which it would be difficult to hide by any means, natural or artificial.

FIG. 341.—*Case* 8. Note prominence in neck.

Case 7.—A girl of 6 had a tumour in the usual position.

OPERATION.—The cyst was removed through a transverse incision. It recurred within three weeks.

Case 8 (*Figs.* 341 and 342).—A man, age 28. He complained that the swelling in the neck was increasing in size. It interfered with deglutition, slipping up behind his collar during the act of swallowing. He had 'neurasthenia' one year ago.

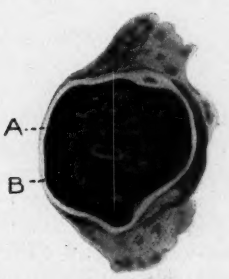


FIG. 342.—*Case* 8. Cyst excised. Note colloid among the blood. A, Colloid; B, Blood. (*Natural size.*)

At the time of the operation there was no sign of exophthalmic goitre.

OPERATION.—Under local anæsthesia a transverse incision was made over the lowest part of the tumour, so that the subsequent scar should be out of sight. The tumour was connected with the hyoid bone by a thin fibrous band, and to the isthmus of the thyroid gland by a broader band of a similar nature. It was easily removed, and a subcuticular suture finished the operation. The cyst when cut across was found to consist of a firm capsule containing a mixture of translucent colloid and blood. The blood probably resulted from pressure by the collar, and may explain its rapid increase. Chemical examination demonstrated the presence of iodine. Two months later the scar was barely visible, and there was no recurrence of the cyst.

FIG. 343.—*Case* 9. Observe cyst in neck.

Case 9 (*Fig.* 343).—A boy, age 2. A small superficial cyst was present between the thyroid cartilage and hyoid bone. It was noticed three months ago.

OPERATION.—A transverse incision was made and the cyst removed. It was adherent above to the thyroid cartilage. There was no return in three months, and no keloid had developed.

Case 10 (*Fig.* 344).—A wasted baby of 5 weeks was admitted on account of difficulty in swallowing. In hospital he had several attacks of dyspnœa, one of which ended fatally five days after admission.

POST-MORTEM EXAMINATION.—A cyst was found at the foramen cæcum of the tongue. There was some œdema of the glottis, and one lung was in a condition of indurated pneumonia.

MICROSCOPIC EXAMINATION.—One sagittal section through the back of the tongue, epiglottis, and part of the larynx. A large cyst in the hinder part of the tongue. It has an epithelial lining, for the most part of single cells, but occasionally this kind is replaced by flattened cells in two or more layers; in one or two places an appearance of several layers, but this may be due to obliquity of section. No colloid or other contents of the cyst are to be found, a

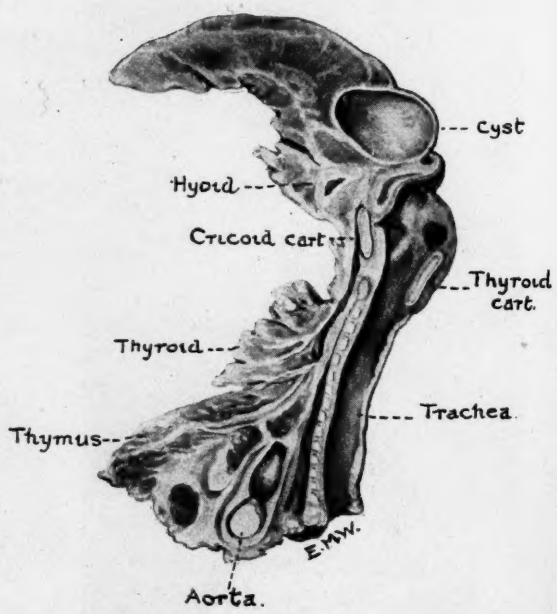


FIG. 344.—*Case* 10. Tongue and larynx showing cyst. Post-mortem specimen. (*Natural size.*)

fluid having probably escaped after hardening. A loose, thin, fibrous wall surrounds the cyst. This is compressed on its upper surface, as is also the submucous layer here, so that the cyst is only separated from the surface by a very thin wall. Groups of glands lie in front of the cyst, below the submucous layer, but none is to be found between it and the surface. No thyroid formation, or prolongation of cells from it in a downward direction, is to be seen, but the section is probably not a median one.

ADENOMA.

Case 11.—A child of 14 months had a small swelling in the neck, about the level of the hyoid bone, which was increasing in size. There were no symptoms.

Operation.—A transverse incision exposed two small pea-like bodies 3 mm. in diameter, resting on the mylohyoid just above the hyoid bone, one on each side of the raphé. In removing the first a relatively large artery was cut, and it was decided to leave the second one, lest it should prove to be the only functioning thyroid. On section the excised tumour proved to be solid ; its character was determined by microscopic examination. Subsequent progress uneventful.

Microscopical Examination.—A typical adenoma was found.

DIFFERENTIAL DIAGNOSIS.

The position of these cysts and fistulæ and their movement on swallowing are so definite that their diagnosis is comparatively easy. The following conditions, however, give rise to some confusion :—

1. Suprahyoid lymph-glands situated on the mylohyoid, as seen in the pea-like bodies of *Case* 11 ; especially if these suppurate or become tuberculous.

2. Sequestration dermoids have been reported here, as elsewhere, in the mid-line of the body.

3. The infrahyoid bursa. This is rarely the seat of pathological processes.

OPERATIVE RESULTS.

Undoubtedly, incomplete removal is the most common cause of failure, but the inexact knowledge of the thyroglossal tract and its relationship to the hyoid bone account for some of the poor results of operation. It has been observed behind, in front, and in some cases passing through a foramen in the bone, and many cases are recorded where a cure was effected only after resection of the middle portion of the bone. This led to the supposition that the tract originally passed deep to the hyoid bone in its course from the tongue to the neck. From embryological findings this is not possible. *Fig.* 337 shows the sinus to be running upwards to the anterior surface of the hyoid bone. Resection of part of this bone need not therefore be a routine procedure, it being possible to dissect it out from behind the hyoid bone as was done in *Case* 5. Another reason for unsatisfactory results is that the fistulæ always become infected, and frequently attention is first drawn to the cysts when suppuration sets in, and they become tender. Again, cells of an embryonic character, but of varying types, are met with lining the cysts or fistulæ, adenomata being rarely met with. The nature of the cell-lining may possibly influence the result. Columnar-ciliated and squamous cells are all found, and are probably sequestrated from the buccal cavity. The impression given by microscopic examination of these cases points to the general existence of

rudimentary thyroid structure lying embedded in fibrous tissue tending to form vesicles by the cavities running together (J. E. F.). This would be instrumental in the causation of recurrences. It is curious that, in spite of the situation of the epithelium, and the number of cases recorded, we have been unable to find in the literature an example of carcinoma arising in the tract. The type of cell found would be of interest.

At operation a horizontal incision in the line of the natural crease of the skin of the neck should be adopted whenever possible. By this means the resulting scar is practically hidden, and as the operation—especially in females—is often done for cosmetic reasons, this is of real importance. This becomes very obvious when *Case* 5 is compared with *Case* 6.

There is no record of the employment of X rays in the treatment of these cases, but it is suggested that a suitable dose before and after operation might prevent recurrence by killing off any bacteria, and possibly inhibiting the growth of the cells.

THE SECRETION OF THE THYROID GLAND.

In *Case* 1 it was seen that the discharge from the sinus was increased during the active period of digestion; this fact had already been noted by several observers. Thus Spencer[2] states that " if the cyst is simply punctured, or incompletely removed, a sinus persists, which discharges mucus as the patient swallows, although there is no connection with the mouth or pharynx ". The explanation is still to seek; it may be simply mechanical, or of great significance. The acts of mastication and deglutition may cause the expression of a larger amount of fluid already contained in the sinus, or it may point to the possibility that the sinus is itself a secreting organ, functioning in sympathy with the other organs concerned in digestion.

An attempt was made to find a simple method of testing for the presence of iodine in the contents of the cysts on the lines laid down by Andrew Hunter.[3] The principle involved was the conversion of all iodine in organic combination into iodate by incinerating with an alkaline fusion mixture. Free iodine was liberated from the iodate by the addition of excess hydrochloric acid and potassium iodide, and its presence shown by the blue colour with starch. The reactions are :—

1. $NaIO_3 + HCl = HIO_3 + NaCl$
2. $HIO_3 + 5KI + 5HCl = 5KCl + 3H_2O + 3I$

A fusion mixture of potassium and sodium carbonates and potassium nitrate was prepared. The contents of the cyst were evaporated and powdered. One grm. of the powder was mixed with 20 grm. fusion mixture and placed in a nickel crucible, when the whole was heated strongly. At first it charred, then ignited, finally turning white. The residue was lixiviated with water; enough 10 per cent hydrochloric acid was added to neutralize the carbonates and leave an acid solution, and with it 1 c.c. of 10 per cent potassium iodide. A drop of 10 per cent starch was added, a blue colour indicating the presence of iodine. A quantitative estimation carried out exactly as described by A. Hunter would have given better information, as control experiments showed the presence of iodine in other tissues. McCarrison[4] has found iodine in most

tissues, but whereas it never exceeds 0·001 per cent elsewhere, its minimum in the healthy thyroid is 0·01 per cent. No other tissue can fix iodine in organic combination.

The staining reaction of the contents of the cysts and the naked-eye appearance of the cyst in *Case* 8 point to their containing thyroid secretion. McCarrison has shown that " the staining capacity of the colloid varies with the phase of activity of the gland. Fresh secretion stains feebly, old collections readily, varying in direct proportion to its iodine content ".

The intimate association of the thyroid gland with the digestive tract is well known. A most important observation is that of Kendall :[5] " The administration of thyroid hormone appears to have no direct action on the pulse. If amino-acids are injected simultaneously, the pulse-rate is enormously affected, and even death may result from the very great metabolism going on in the animal. It appears very probable that the thyroid's hormone manifests its activity in some way with amino-acids ", i.e., with protein ingestion.

Diseased conditions of the intestines are reflected in pathological changes in the thyroid, and vice versa. Thus McCarrison states : " Intestinal stasis may be due primarily to subthyroidism, and this is especially the case where the thyroid defect is congenital or acquired in early life. If due to other causes, the stasis, once established, may lead to subthyroidism by the action of the gland on the production of bacterial growth in the static bowel, or the abnormal process of digestion may interfere with the efficient elaboration of the thyroid secretion, thus reducing the physiological activity. Thus a vicious circle is set up ". The same author has shown that it is affections of the upper intestinal tract which are responsible for pathological changes in the thyroid. " A condition of hypo-acidity of the gastric contents is the rule in exophthalmic goitre, and this is, in itself, favourable to the infection of the upper part of the alimentary tract by micro-organisms of disease. The frequency with which such disorders of the tract precede the onset of Graves' disease is becoming increasingly evident. Dilatation of the stomach is frequent, as is an onset with vomiting and diarrhœa ". Simple goitre has been proved fairly conclusively to be due to fæcal contamination of food or water (McCarrison).

The successful use of intestinal antiseptics was suggested to McCarrison " by the fact that in certain animals (e.g., amphioxus) the thyroid is connected with the upper end of the intestinal canal by a duct, and probably exercises a destructive action on bacteria entering the tube ". It is an undoubted fact that thyroid medication by the mouth is much more satisfactory than that of any of the other ductless glands.

Carlson, Dooks, and McKie find that the exhibition of toxic doses of thyroid substance in animals causes gastro-enteritis and diarrhœa.

It has been shown that parathyroids also play a part in digestion, in that they neutralize guanidine, a substance formed during purin metabolism.[6] If injected in large doses, guanidine is capable of causing the nervous syndrome-complex of tetany in normal animals. The serum of dogs from which the thyroid has been removed acts upon the muscles of the frog similarly to weak solutions of guanidine.

An interesting observation is that "in exophthalmic goitre, where the gland is greatly stimulated, the activity of the salivary glands is usually increased, and salivation may be paroxysmal".[4]

Giordana and Caylor are uncertain whether or not the involution changes following ligation of the thyroid vessels in exophthalmic goitre are attributable to the lessened blood-supply or to the division of the sympathetic nerves accompanying these vessels.

The main points arising out of these facts give rise to the suggestion that possibly both the sinus and the main thyroid have an innervation identical with that of the salivary glands, the whole acting in sympathy. If this should prove true, new paths for research are opened up in the consideration of the treatment, medical and surgical, of affections of the thyroid, e.g., exophthalmic goitre. Different foodstuffs may have different effects on the thyroid, as Pavlov[7] has shown in the case of the salivary glands. Instead of ligaturing arteries, attention may possibly be more usefully paid to the peri-arterial nerve plexus.

SUMMARY.

1. Division of the hyoid is rarely necessary if the dissection is carried up well behind the bone.

2. A horizontal incision, if possible, along a natural crease of the neck gives the best results as regards the subsequent scar.

3. A suggestion is put forward that the thyroglossal fistula is in direct anatomical relation with the thyroid gland, and that, *pari passu* with that of the salivary glands, its activity is increased during digestion.

Thanks are due to the honorary surgeons of the General Infirmary at Leeds for permission to use their cases. For the radiograms I am indebted to the X-ray department at Leeds, and for *Figs.* 342 and 344 to Miss E. M. Wright. [A. P. B.]

REFERENCES.

[1] BERTWISTLE, A. P., *A Descriptive Atlas of Radiographs of the Bones and Joints*, 1924: John Wright & Sons Ltd., 192, 193.
[2] SPENCER, W. G., Gask and Wilson's *Surgery*, 709.
[3] HUNTER, A., "Determination of Small Quantities of Iodine, with Special Reference to the Iodine Content of the Thyroid Gland", *Jour. of Biol. Chem.*, 1909—10, vii.
[4] MCCARRISON, R., *The Thyroid Gland*, 16, 17, 45, 52, 187, 220, 226, 227, etc.
[5] KENDALL, E. C., *Boston Med. and Surg. Jour.*, 1916, Oct., clxxv, No. 16.
[6] PATON and FINDLAY, *Quart. Jour. Exper. Physiol.*, 1917, x, 203.
[7] PAVLOV, *Le Travail des Glandes digestives* (Russian text, St. Petersburg, 1897; French Translation, Paris, 1903).

THYROGLOSSAL CYSTS AND FISTULÆ.

By HAMILTON BAILEY, London.

The gifts of embryology to surgery are manifold. The thyroglossal tract, not the least of these, was certainly one of the first to be generally appreciated.

For this paper it was considered advisable to adhere strictly to the surgical aspects of thyroglossal cysts and fistulæ, and 117 cases have been collected from the records of the London Hospital for this purpose. Whilst in hospital surgical practice thyroglossal cysts are frequently encountered, the actual incidence in the general population is probably exceedingly small, for in 86,000 consecutive patients examined in the Mayo Clinic only 31 cases of thyroglossal cysts were found (Sistrunk). In common with affections of the thyroid gland, thyroglossal cysts and fistulæ are commoner in women. Thus, of the 117 cases reviewed, 75 occurred in females.

THYROGLOSSAL CYSTS.

GENERAL DIAGNOSIS.

The diagnosis of thyroglossal cyst is usually simple. Certain difficulties are met with from time to time; the chief of these results from a peculiar liability of these cysts to infection. If a thyroglossal cyst comes under observation for the first time when thus complicated, its true nature is easily overlooked, and a diagnosis of some inflammatory condition is made, notably an abscess 'en bouton de chemise' connected with a tuberculous pretracheal gland. Less frequently, when fluctuation cannot be obtained because the cyst is tense and small, an enlarged gland enters the question. Again, in the middle line a simple epidermoid cyst is a difficult but an unimportant differential diagnosis. In this connection it should be mentioned that a thyroglossal cyst quite frequently moves upwards on fully protruding the tongue, and this sign may serve to distinguish it from an epidermoid cyst. The translucency test is worth trying, but as only about

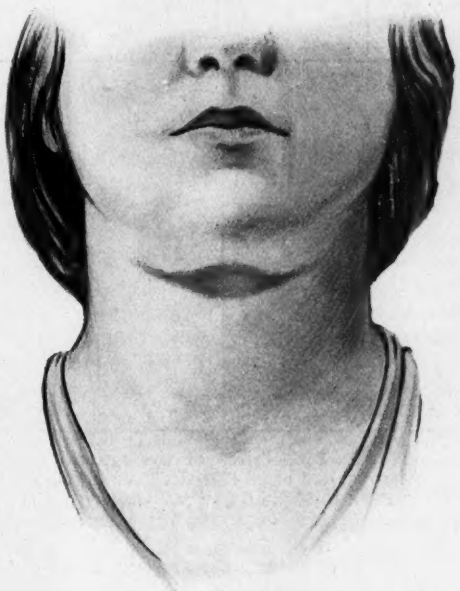


Fig. 345.—Acute inflammation in a thyroglossal cyst. This case illustrates the tendency of the accompanying cellulitis to spread laterally and surround the neck like a necklace. The cyst and the tract were later enucleated. (*Sir Hugh Rigby's case.*)

5 per cent of thyroglossal cysts are translucent, no importance can be attached to a negative result.

Lastly, a diagnosis of thyroglossal cyst is never complete without definitely determining the presence of a thyroid gland in its normal position, for an ectopic thyroid has probably many times been unwittingly removed. Not infrequently this thyroid is the only thyroid, for, as Spencer[1] states, "The persistence of thyroid tissue in the course of the thyroglossal tract stands as an inverse relationship to the normal development of the normal thyroid gland".

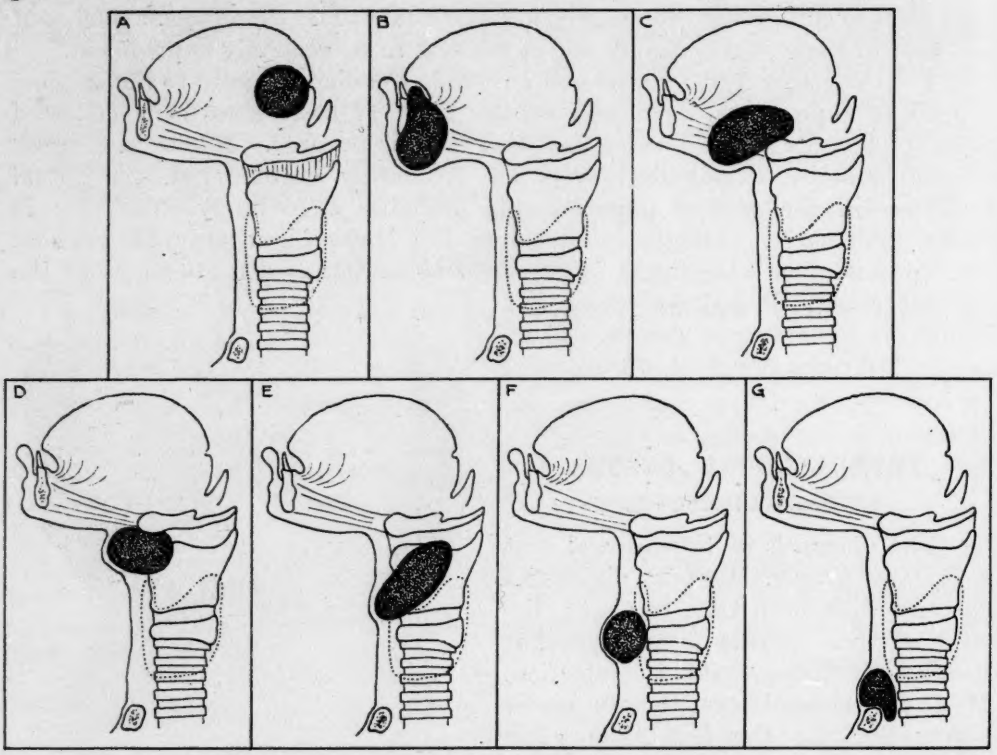


FIG. 346.—Sites of thyroglossal cysts. A, Beneath foramen cæcum (two cases at necropsy), 3 cases; B, In floor of mouth protruding beneath the chin, 6; C, Suprahyoid, 12; D, Subhyoid, 37; E, On thyroid cartilage, or membrane, 23; F, At level of cricoid cartilage, 7; G, In the suprasternal notch, 4.

As a dresser, I assisted in the out-patient clinic to remove a small lump from the mid-line of a child's neck just below the hyoid bone. The histological report was normal thyroid. The girl is now attending hospital with myxœdema. Cases of this kind simulating thyroglossal cysts have been reported.

Sites of the Cyst.—In 92 cases the site was recorded (*see Fig.* 346).

THE ESPECIAL CLINICAL FEATURES OF THYROGLOSSAL CYSTS IN VARIOUS SITUATIONS.

1. **At the Base of the Tongue beneath the Foramen Cæcum.**—This is the most infrequent site for a thyroglossal cyst to develop, and there was in this series but a solitary example diagnosed and treated. It occurred in an

infant of 10 weeks. The records of the London Hospital Pathological Institute afford two further instances, both of which were seen at necropsies upon young subjects. Rare as this situation is for a thyroglossal cyst, it is the situation *par excellence* for an ectopic thyroid.

2. In the Floor of the Mouth.—(*Fig.* 347).

a. Many 'lingual dermoids' are almost certainly squamous-lined thyroglossal cysts; at the present time there seems to be very little dissension upon this point. The sebaceous material within the cyst gives the swelling that yellow hue by which it is so readily recognized. In common with all other thyroglossal cysts the lingual dermoid is liable to attacks of acute inflammation.

FIG. 347.—Sublingual dermoid. An extension of the cyst can be felt beneath the chin. (*Mr. Hugh Lett's case.*)

Case 1.—A boy of 9 was admitted to the London Hospital with considerable dyspnœa and extensive œdema of the tongue. Temperature 103°. The cavity of the mouth was entirely obliterated by a cystic swelling protruding from beneath the œdematous tongue. There was considerable swelling beneath the chin. The cyst was incised, and a large quantiy of material 'like curds' was evacuated. Three weeks later the cyst was excised through the floor of the mouth; the cavity was packed.

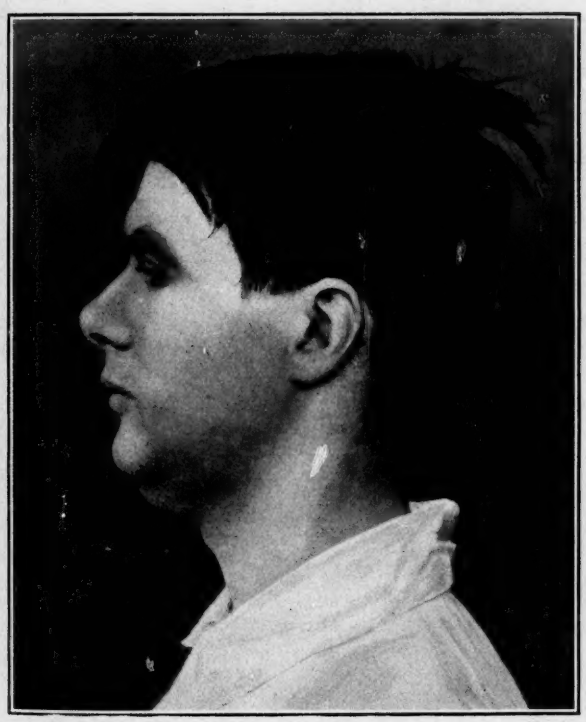


FIG. 348.—Suprahyoid thyroglossal cyst. The patient has previously received surgical treatment for a 'ranula'. (*Mr. Sherren's case.*)

b. Relationship to Ranula.—'Ranula' is a conglomerate term which still embraces several distinct conditions. With the advent of exact pathology, one by one various clinical entities have been recognized and withdrawn from the umbrage of ranula, a term which Hippocrates applied to a cystic swelling in the floor of the mouth, owing, it is believed, to the likeness such a swelling bore to the belly of a little frog.

At the present time it would seem desirable to reserve the term 'ranula' for a cystic degeneration of the sublingual gland or the gland of Blandin and Nuhn. Such cysts of salivary glandular origin, when they have attained considerable dimensions, may extend under the frænum linguæ to the opposite side, but the bulk of the swelling is always manifestly unilateral. Some truly median ranulæ and many so-called ' deep ranulæ ' (i.e., those with an extension beneath the symphysis menti) are in reality the mucous variety of thyroglossal cyst. Von Fleischmann, recognizing that this type of case was unlike an ordinary ranula, attributed the condition to an enlargement of a bursa between the two geniohyoglossi muscles. Later investigation showed that there was no anatomical or pathological evidence of such a bursa.

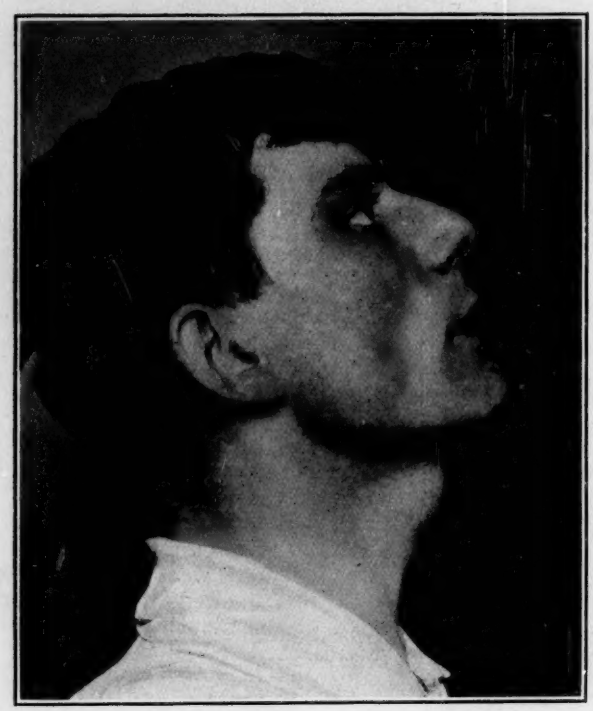


FIG. 349.—Subhyoid thyroglossal cyst. Present since early childhood, it has lately grown larger. (*Mr. Walton's case.*)

3. **Suprahyoid.**—A thyroglossal cyst situated immediately above the hyoid (*Fig.* 348) presents no peculiar differential difficulties.

4. **Subhyoid.**—This is the commonest site for a thyroglossal cyst (*Fig.* 349). When the patient swallows, sometimes the cyst disappears beneath the hyoid bone and remains temporarily hidden there.

A case of this kind was recently observed in a girl of 23. By asking the patient to open her mouth and fully extend the head, it was found that the cyst, which was the size of a walnut, became evident. When a normal attitude was again adopted and the patient swallowed, the cyst disappeared from view with an audible 'click'.

With regard to differential diagnosis, there appears to be very little anatomical or pathological evidence of the existence of a subhyoid bursa. There is no case of an enlarged subhyoid bursa in the records of the London Hospital. Such a condition is probably as mythical as Fleischmann's bursa.

5. **At the Level of the Thyroid Cartilages.**—It is often said that a swelling cannot be a thyroglossal cyst unless it is strictly in the middle line. The exception to this rule is the cyst in relationship to the ala of the thyroid cartilage. The thyroid cartilage, shaped like the prow of a ship, in the course of its development sweeps to one side (usually the left) the thyroglossal

tract. Thus the levator glandulæ thyroideæ in dissecting-room subjects is usually found on the left side of the thyroid cartilage; we may therefore expect a thyroglossal cyst at this level usually to be situated on the left side, and this is so (*Fig.* 350),. Of the 23 cases belonging to this group it is recorded that the cyst was on the right side of the middle line in 6 and on the left in 9. This deviation often makes the differential diagnosis between an inflamed thyroglossal cyst and a tuberculous gland the more difficult.

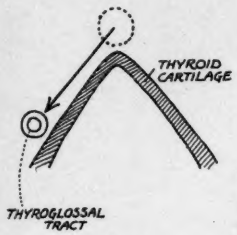


FIG. 350.

6. **At the Level of the Cricoid Cartilages.**—At this level a thyroglossal cyst tends to assume the middle line once more, but it is not very unusual to observe some slight departure from the median plane.

It is here that differential diagnosis has particularly to be made from a solitary thyroid adenoma, and the nearer the cyst is to the isthmus of the thyroid, the more difficult does this differential diagnosis become.

7. **In the Suprasternal Fossæ.**—The thyroglossal tract can very occasionally be traced downwards far below the limits of the thyroid isthmus. It is therefore submitted that some of the 'dermoids' situated in the suprasternal fossa may have their origin in the thyroglossal tract. Such a hypothesis is strengthened by the fact that the lining epithelium of the cysts removed from Burn's space is occasionally columnar. The possibility that these are thymic duct remnants cannot be denied. A cyst in this situation is difficult to examine, but is usually thrown into prominence by fully extending the neck (*Fig.* 351).

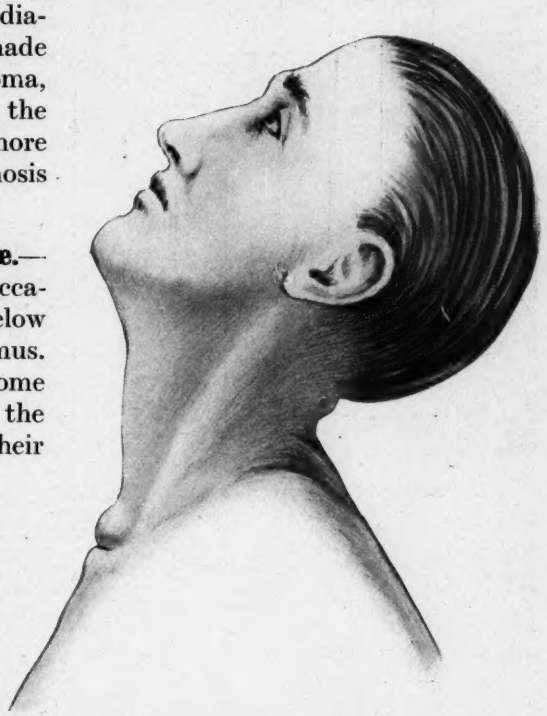


FIG. 351.—A cyst in the suprasternal notch. It is difficult to feel, and cannot be seen unless the patient fully extends his neck. (*Mr. Russell Howard's case.*)

A differential diagnosis of the cyst in this situation in an adult rests particularly from a retrosternal goitre, whilst in a child an enlarged thymus should receive consideration.

Case 2.—Bootmaker, age 48. Since childhood has had a lump in the neck. Some discomfort whilst singing was the only symptom until six months after a blow on the throat, when the lump increased in size and became painful. The cyst was excised from Burn's space, and at operation an abnormal depression was observed on the superior aspect of the manubrium sterni.

HISTOLOGICAL REPORT.—Inflamed cyst, lined by columnar epithelium.

AGE OF FIRST APPEARANCE.

In contradistinction to branchial cysts[2], which most frequently appear in early *adult* life, thyroglossal cysts are commonly met with in babies, and, as *Fig.* 352 shows, the majority of the cases reviewed occurred in infants, or the history of the trouble dated back to very tender years. In spite of this fact it will be noticed that 19 cases had no signs until after the twentieth year.

To recapitulate: thyroglossal cysts sometimes, and branchial cysts generally, make their first appearance after the second decade of life.

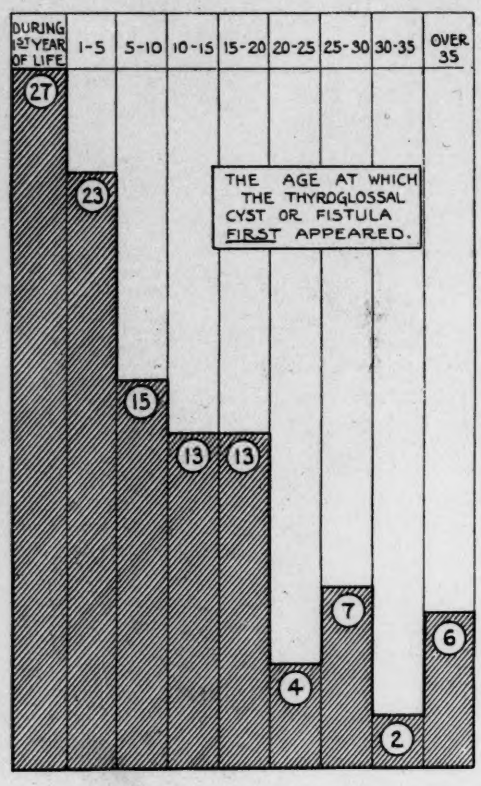


FIG. 352.—Table showing age of appearance of thyroglossal cysts or fistulæ.

It is truly mysterious that these embryological remnants, after having slumbered in the neck for years—perhaps until the prime of life or even later—should suddenly, or comparatively suddenly, burst into activity and disclose their presence in the shape of a cyst. As far as I am aware, no explanation has been recorded which accounts for the phenomenon.

The case of thyroglossal cyst might be explained by a patent thyroglossal duct. If such a duct, opening into the mouth at the foramen cæcum, became for some reason or another occluded, then a thyroglossal cyst might be expected to result. Nevertheless, such a theory postulates a patent thyroglossal duct opening into the mouths of a certain percentage of normal individuals, for it would be unreasonable to suppose that it always became occluded. The available evidence,[3] such as it is, does not substantiate this proviso. Furthermore, this theory entirely fails to explain the parallel case of branchial cyst, which in the vast majority of cases is quite unconnected with the pharynx.

The following explanation, which holds good for both these embryological remnants, is submitted: Thyroglossal cysts and branchial cysts are not infrequently surrounded by lymphadenoid tissue. In common with the neighbouring lymphatic system, this from time to time becomes the seat of bacterial invasion. If this is so, then the lining epithelium is liable to become involved, and reacts to the infection in just the same manner as other epithelial surfaces (notably the nasal mucous membrane) by pouring forth secretion.

Some evidence in favour of this theory is as follows :—

1. Four cases of thyroglossal cyst appearing in adolescent or adult life commenced with tonsillitis or pharyngitis *for which the patient had received treatment.* Several more gave a history of sore throats which may or may not be relevant.

2. A baby admitted with an infected foot developed a thyroglossal cyst, which suppurated.

3. There is overwhelming proof, both clinical and histological, to show that thyroglossal cysts are frequently the seat of inflammation. Furthermore, the wall of branchial cysts upon histological examination are often found to be chronically inflamed.

THYROGLOSSAL FISTULA.

Raymond Johnson[4] first pointed out that this is rarely if ever a congenital condition. Thyroglossal fistula is the result of rupture of a thyroglossal cyst.

FIG. 353.—True congenital thyroglossal fistula. The patient's father and mother noticed this condition on the day of birth. No operation has at any time been performed. In the upper part of the scar is a fistula B, which passes upwards, and, associated with this, there is a small subhyoid thyroglossal cyst. There is also a fistula D, passing downwards behind the manubrium. The intervening portion between B and D is made up of thin scar tissue, and represents the posterior wall of that portion of the thyroglossal tract between the two fistulæ. (*Sir Hugh Rigby's case.*)

Of the 31 cases of fistula included in this series there is only one which may be the exception to this now well-established rule. [Since writing this another case of true congenital fistula has come under observation (*Fig.* 353).]

Case 3.—Female, age 18 months. Since birth the mother noticed a tag of skin in the middle line of the baby's neck. Examination showed a small redundant flap of skin hanging down in the middle line ; on lifting this up, the orifice of a

sinus was displayed. A probe could be passed into the sinus in an upward direction for ¾ in. and downward for ½ in. There was no discharge at the time of this examination. The sinus was dissected out.

In the remaining 30 cases there is no question of the fistula being congenital; it was acquired at various times during life between the third week and the thirty-first year. Suppuration in a thyroglossal cyst, favouring as it does both spontaneous rupture and treatment by incision, plays a large part in the production of the fistula. Nevertheless, the commonest direct cause is some form of surgical intervention, for in 17 out of the 31 cases there was a clear history of the discharge commencing after an operation upon the neck. The exact nature of this operation was not always clear, and it includes both incision and excision of a thyroglossal cyst, but in the large majority of cases it is quite evident that it was the latter. Kyle[8] suggests that, in order to ascertain the extent of a thyroglossal fistula, lysol should be injected into the external orifice: if the tract is complete, the patient tastes this substance. So exceptional is a complete thyroglossal tract opening on the one hand at the foramen cæcum and on the other externally that one wonders if this test has ever given a positive result.

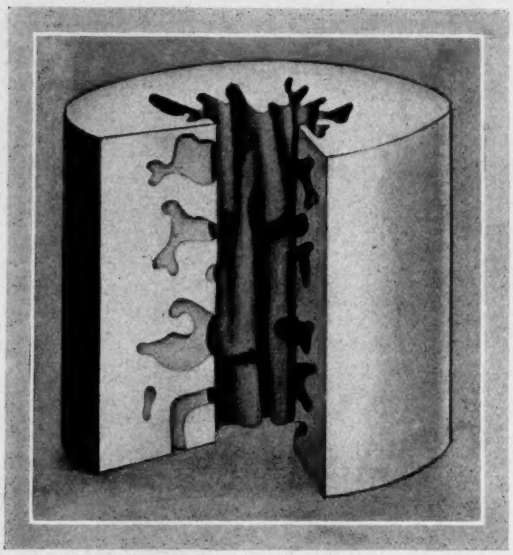


FIG. 354.—Seelig's specimen of a thyroglossal fistula reconstructed in wax from serial sections. Note the numerous lateral diverticula from the main channel. (*Redrawn from Seelig.*)

It would certainly be very advantageous, especially in recurrent cases, to be able to ascertain before operation the extent and ramifications of the fistula (*Fig.* 354). Doubtless, this information could often be obtained by the injection of the tract with sodium bromide and subsequent radiography, and it seems particularly desirable that this method should be tried in all cases.

The amount of excretion discharged varies within wide limits. Sometimes it is continuous, and one male patient's chief complaint was that his collars were spoilt by it. At other times the discharge is intermittent, and in some of the latter cases a cyst was formed in the thyroglossal tract during the free interval. In uncomplicated cases when the excretion is copious it tends to be thin and watery, but in the intermittent cases it may be either viscid like white of egg or opaque and pus-like from contained cholesterol.

In fistulæ of long standing which originated during infancy the orifice is situated in the neighbourhood of the cricoid cartilage, tending to be rather below than above this structure. The scar surrounding these long-standing fistulæ always has a peculiar crescentic appearance, which is doubtless due to the uneven rates of growth of the thyroglossal tract and the

rest of the neck, causing tension at the point where the fistula pierces the integument.

From time to time cases are seen in which this peculiar scar, like an extinct volcano, alone is present. *Fig.* 355 shows such a scar in a middle-aged man who came to hospital for an injury to his wrist. There was no history of a discharge from the neck. The patient suggested the scar was a 'birth mark'.

The foregoing remarks are applied to external thyroglossal fistulæ, for in this somewhat extensive series there is no case of the fistula opening into the mouth at the foramen cæcum. Freer[5] has observed an example of the latter in which the discharge of a thyroglossa fistula into the postlingual space caused obstinate cough; whilst New[6] has injected bismuth paste into such a sinus, demonstrating its extent radiologically.

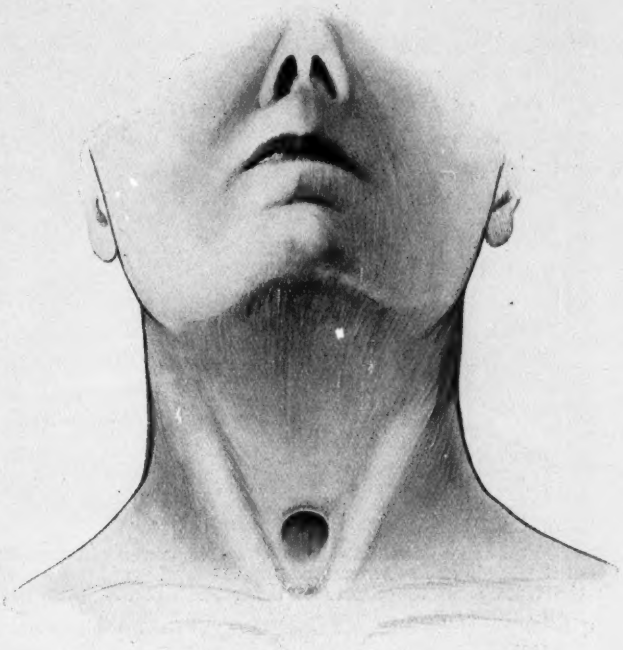


FIG. 355.—A thyroglossal scar, without fistula. The patient came to hospital with an injury to his wrist. (*Mr. Walton's case.*)

TREATMENT.

The practical importance of this little subject is liable to be overlooked until we examine the results of removal of thyroglossal cysts and fistulæ. Without the advantage of any special investigation by 'following up' cases, 26 per cent of the total cases reviewed were proved by the records to be recurrent. A proportion of these have been operated upon more than twice.

It is necessary to make it perfectly clear that by 'recurrent' is understood both cases which were initially operated upon at the London Hospital and those which originally received similar treatment elsewhere. In view of the probable mutual exchange of failures amongst the hospitals, 26 per cent should represent a mean average.

Two methods of treatment have given such unsatisfactory results that they have now been largely discarded: (1) The simple dissection of a cyst, combined with proximal ligature of its thyroglossal pedicle; and (2) Treatment of thyroglossal fistulæ by means of scraping and cautery.

When an operation for a thyroglossal cyst is undertaken, the goal in view is to extirpate once and for all the cyst and every vestige of the tract. The very great majority of thyroglossal cysts are situated in some part of the

infrahyoid portion tract. The extirpation of this portion of the tract is comparatively easy; there is nothing to disturb the even tenor of the dissection as far as the hyoid bone. It is here that trouble begins and the conflicting views of leading anatomists* upon the relationship of the tract to hyoid are forcibly impressed upon the operator. Often it is here that the thyroglossal pedicle, already considerably attenuated, seems to dwindle into mere shadow, and the dissection has, perforce, to be terminated. The operation is sometimes considerably facilitated by simple division[9] of the hyoid bone, but even this step does not necessarily secure success.

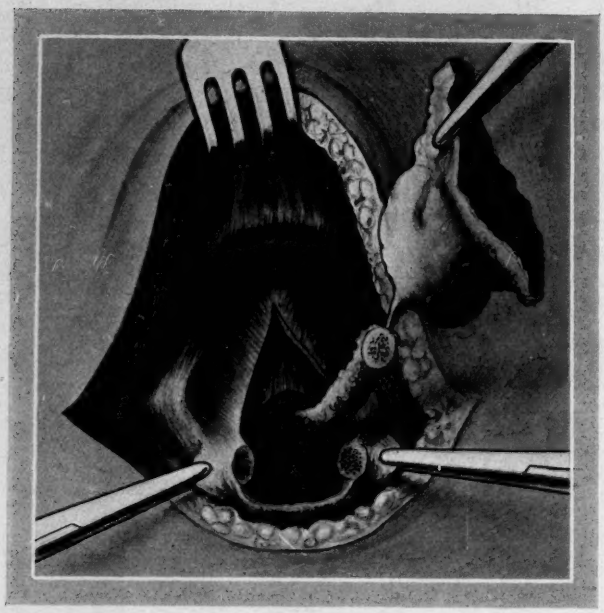


FIG. 356.—*Sistrunk's operation.* The centre of the body of the hyoid has been resected and the suprahyoid portion of the tract is in the course of being enucleated. (*Redrawn from Sistrunk.*)

Sistrunk's Operation.—Sistrunk,[7] realizing the unsatisfactory result of many of these cases, has devised a technique which circumvents the difficulties encountered in the hyoid region and eradicates the supra- as well as the infrahyoid portion of the thyroglossal tract.

The operation is usually carried out through a transverse incision about the level of the hyoid bone (*Fig.* 356). The thyroglossal tract having been dissected as far as the body of the hyoid, the attachments of muscles in the immediate vicinity of this structure are separated, and $\frac{1}{4}$ in. of the centre of the bone is resected. Then, without attempting to isolate the duct, the tissues are 'cored' through until the foramen cæcum is reached, allowing about $\frac{1}{3}$ in. on every side of the duct. In order that this procedure may be carried out it is

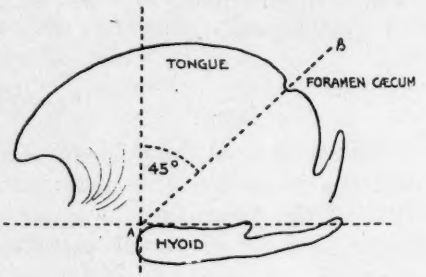


FIG. 357.—*Sistrunk's operation.* The suprahyoid portion of the thyroglossal tract is eradicated by 'coring out' the tissues along the line A-B.

* 1. The thyroglossal tract passes through the body of the hyoid (Professor Parsons).
2. The thyroglossal tract passes in front of the body of the hyoid (Professor Frazer).
3. The hyoid bone develops in the course of, and interrupts the continuity of, the thyroglossal tract (Sir Arthur Keith).

necessary to know the exact course of the suprahyoid portion of the thyroglossal tract. If two intersecting lines are drawn, one horizontal and one perpendicular to the superior border of the body of the hyoid bone, then the foramen cæcum lies upwards and backwards from the hyoid at an angle of 45°, and it is along this line that the dissection proceeds until the cavity of the mouth is reached (*Fig.* 357). The dissection now completed, the opening in the mouth is closed. Several sutures are used to draw the geniohyoglossi muscles together. The tissues around the cut ends of the hyoid bone are brought together with chromic gut sutures in such a manner as to approximate the cut edges of the bone. A small rubber tube is inserted from this point to the skin incision, which is now closed. The dissection removes *en bloc*:—

1. The thyroglossal tract.
2. The centre of the body of the hyoid bone.
3. Portion of the median raphé of the mylohyoid.
4. Portion of the geniohyoglossi muscles.
5. The foramen cæcum.

REFERENCES.

[1] Spencer, W. G., *Lancet*, 1914, i, 522.
[2] Bailey, Hamilton, *Brit. Jour. Surg.*, 1923, x, 565.
[3] Kanthack, A. A., *Jour. Anat. and Physiol.*, 1890, 157.
[4] Johnson, Raymond, *Lancet*, 1890, May 10.
[5] Freer, Otto, *Trans. Amer. Laryngol. Assoc.*, 1918, xl, 239.
[6] New, G. B., *Surg. Clin. N. Amer.*, 1924, iv, 521.
[7] Sistrunk, W. E., *Ann. of Surg.*, 1920, lvi, 121 ; *Surg. Clin. N. Amer.*, 1921, i, 1509.
[8] Kyle, J. J., *Laryngoscope*, 1905, xv, 880.
[9] Durham, H. E., *Proc. Roy. Soc. Med.*, 1893, 82.
Bland-Sutton, Sir John, *Tumours*, 558–65 and 529.
Grant, Andrew, *Proc. Roy. Soc. Med.* (Laryngol. Sec.), 1908–9, ii, 6.
Seelig, M. G., *Surg. Gynecol. and Obst.*, 1907, iv, 623.

A LARGE CAVERNOUS HÆMANGIOMA OF THE LEFT LOBE OF THE LIVER CAUSING OBSTRUCTION TO THE CARDIAC ORIFICE OF THE STOMACH.

By CECIL P. G. WAKELEY, London.

Although cavernous angiomata are the commonest innocent tumours met with in the liver, they are most frequently multiple and of small size; and even if they grow to a considerable size they rarely give rise to symptoms. It is only when the capsule of the tumour becomes converted into fibrous tissue that a large angioma can be easily palpated per abdomen, for where no capsule is present the cavernous spaces abut directly on the liver tissue, and form a very soft tumour. In the case described below the angioma was about the size of a closed fist and easily palpable; it was mistaken on account of its hardness for a carcinoma of the cardiac orifice of the stomach. The tumour was hard, and on section was found to have a very dense fibrous capsule; it was not compressible even when it was removed from the liver. In fact, the tumour might easily be termed a fibrous angioma on account of its very thick fibrotic capsule. Some authorities still consider that such tumours may be regarded as fibromata containing dilated inter-communicating vascular channels; while others deny that they are of the nature of true tumours, but consider that they are dilatations of the normal blood-spaces. The following is the history of the case:—

Mary L., age 62, complained of difficulty in swallowing which came on insidiously five years prior to her admission to King's College Hospital on May 14, 1924. The patient stated that the food seemed to stick in the 'pit of the stomach' and was only shifted by swallowing some water after each mouthful of food. In April, 1924, all solid foods were vomited and the patient was compelled to take fluid diet. For the year previous to admission she had been steadily losing weight. She had no history of jaundice.

On Examination.—The patient was seen to be thin and worn, with marked retraction of the abdomen. A hard lump about the size of an orange could be palpated in the left hypochondriac region; it was movable on respiration, its surface appearing to be slightly grooved. The upper border of the lump could not be made out. There was no increase in size of the liver and no free fluid in the abdomen. A diagnosis of carcinoma of the lower end of the œsophagus or cardiac end of the stomach was made.

On Admission.—On the patient's entry into hospital a barium meal was administered. She did not vomit the meal, and a skiagram (*Fig.* 358) taken immediately afterwards revealed some opaque meal in the stomach, but the major portion was retained in the lower end of the œsophagus. A skiagram taken two hours later showed the barium in the stomach, duodenum, and small intestine, while there was still a considerable portion in the

lower end of the œsophagus. A skiagram taken after five hours demonstrated that the meal had reached the cæcum; there were also opaque residues in the œsophagus, stomach, and small intestine. The fourth skiagram, taken after eight hours, revealed the majority of the meal in the cæcum and ascending colon, while there were still traces of the meal in the œsophagus, stomach, and small intestine. The fifth skiagram, taken twenty-four hours after the

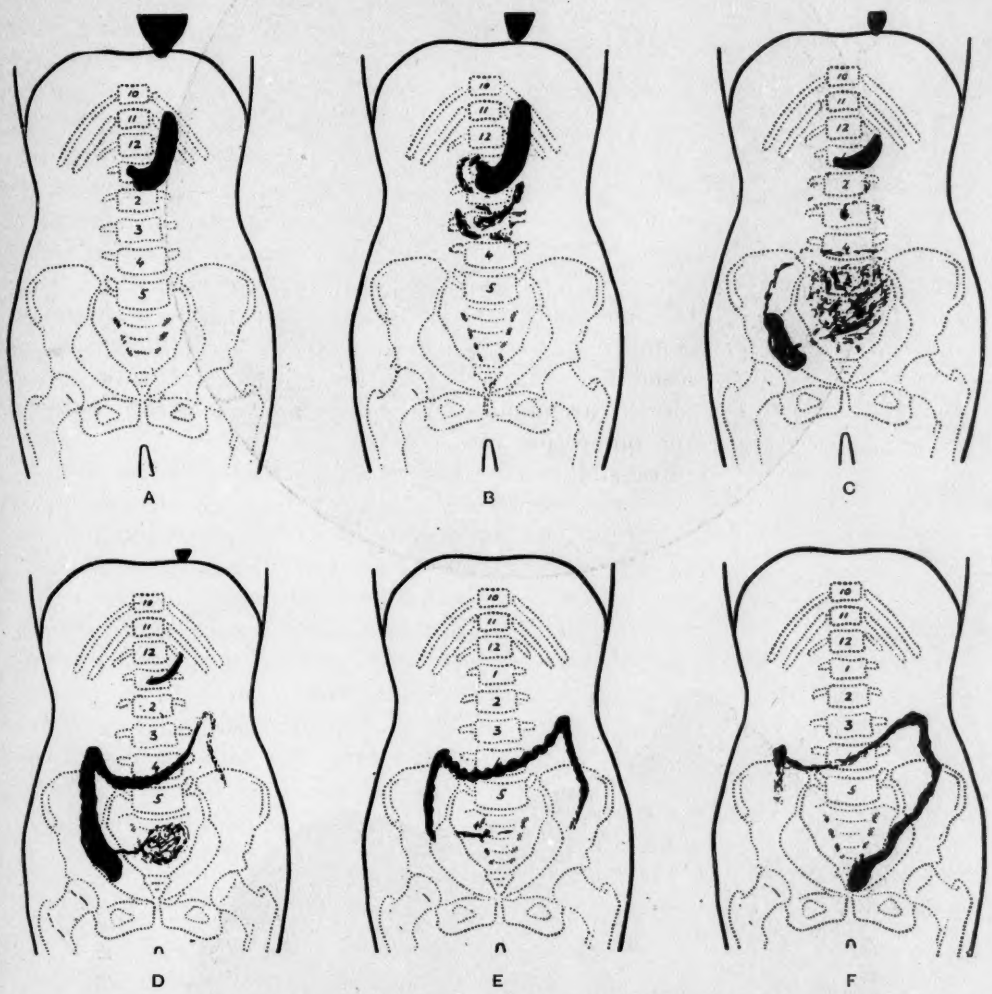


FIG. 358.—Drawings of skiagrams taken after ingestion of barium meal. A, Immediately after; B, After 2 hours; C, After 5 hours; D, After 8 hours; E, After 24 hours; F, After 48 hours.

meal was given, showed that the ascending, transverse, and descending colons contained all the opaque meal; while the sixth skiagram, taken after forty-eight hours, demonstrated that the meal was mainly in the descending and sigmoid colons and the rectum.

OPERATION.—The skiagraphic findings seemed to confirm the clinical diagnosis, and a gastrostomy was proposed to the patient, who willingly

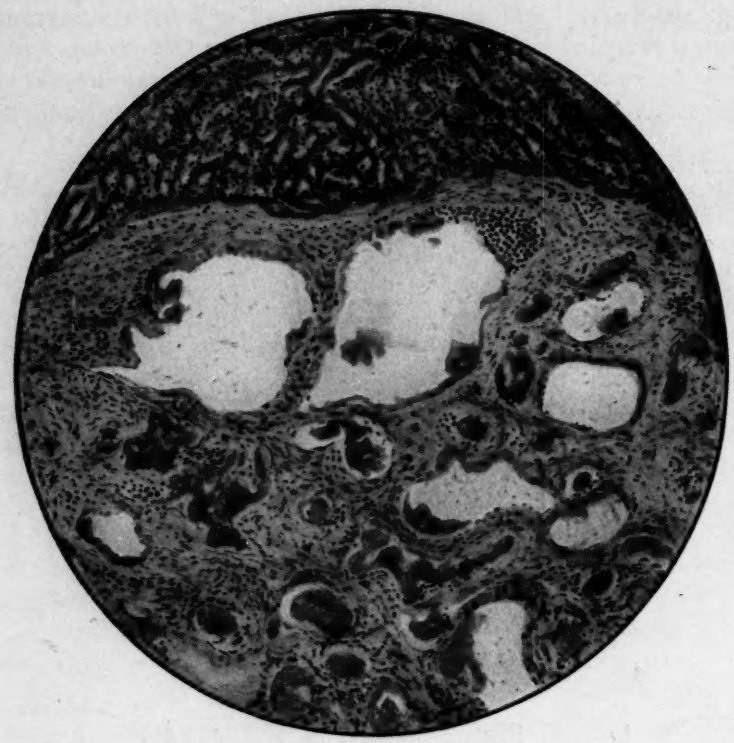


FIG. 360.—Angioma of liver. (× 80.)

FIG. 359.—Angioma of liver. (*Natural size.*)

consented. A laparotomy was performed on May 23, 1924, under gas and oxygen anæsthesia. An incision was made through the left rectus muscle and the peritoneal cavity opened. The stomach was found to be normal, no tumour could be felt, and there was no growth at the lower end of the œsophagus, as a finger could be passed upwards after invaginating the stomach wall. A hard tumour was felt, however, in the extreme end of the left lobe of the liver; it was about the size of a closed fist. Owing to its hardness it was thought to be a malignant neoplasm; a careful examination of the abdomen did not reveal any sign of a primary tumour, so it was decided to remove the growth from the liver by the diathermy cautery. The abdominal viscera were packed off with gauze, and the growth, with half an inch of liver tissue, was removed. Hæmorrhage was not great and was easily controlled by gauze packing, which was left in for forty-eight hours. The abdomen was closed in three layers; a drainage tube was inserted. The gauze packing was removed on the second day under nitrous oxide anæsthesia; the tube was removed three days later. The wound healed soundly.

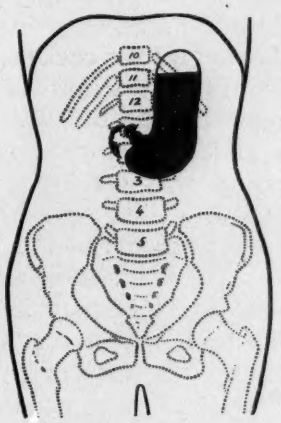


FIG. 361.—Drawing from skiagram taken immediately after barium meal, showing normal stomach outline.

The patient was taking solid food after six days and rapidly put on weight. A barium meal taken three weeks after the operation (*Fig.* 361) revealed a normal stomach with a well-marked duodenal cap; there was no sign of obstruction at the cardia.

The tumour on section (*Fig.* 359) was seen to be surrounded by a definite capsule. The microscopical section (*Fig.* 360), taken through the edge of the tumour so as to include a small piece of liver tissue, shows the typical cavernous angioma met with in the liver; dilated capillary spaces can be seen extending into the liver tissue. The endothelial lining of the spaces is well marked.

A CASE OF UNILATERAL AFFECTION OF THE SKELETON OF UNKNOWN ORIGIN.

By H. A. T. FAIRBANK, London.

Although the curious, and possibly unique, radiographic findings in this case more than warrant its publication, the author has a special object in view in reporting the details. So far he has failed to find anyone who has observed a similar case or who can explain the appearances seen. He hopes that this report may catch the eye of someone who can help him with the diagnosis and who will be good enough to communicate with him. It is most distressing to the parents of the boy that no prognosis can be given.

History.—The patient is 12 years of age. The history is that after an uneventful infancy it was noticed that he always ran awkwardly, turning his toes out, and especially those of the right foot. He got over a stile clumsily with the right leg, and preferred to cross with the left leg leading. Though nothing more definite could be obtained in the way of a history, there was certainly something which drew his parents' attention to the right leg.

Some fifteen months ago he developed rheumatic fever, which affected his heart. Only two or three joints were involved, and these cleared up in three days. After many months' absolute rest in bed he began to get about again, and had recently been passed fit for school, though still unfit to play games. As the awkwardness of the right leg had been rather more noticeable since his long illness, it was thought advisable that the cause of this should be investigated before he returned to school. His medical attendant, Dr. A. Bevan, and Dr. Poynton, who also saw him several times during his long illness, are emphatic as to the absence of any arthritis during that period which could have accounted for the bone changes. There is, moreover, no history of arthritis at any time, nor of any illness which could have a bearing on the case. He has never sustained a fracture. As to the family history, he is the child of apparently healthy parents, who are not cousins. His mother had two healthy children by a previous marriage. There is no history of bone affections in the family.

On Examination.—The patient proved to be a well-developed boy of very fair physique considering his recent illness. He did not limp, complained of no pain, and stepped over a chair readily enough with either leg. The right thigh was thinner than the left by three-quarters of an inch, but this leg was half an inch *longer* than the left. The lengthening was mostly in the femur and only slight in the tibia. The calves were equal in size. The right foot was slightly larger than the left. Both feet were markedly flattened, though supple. The arms were equal in length. Nothing abnormal was discovered in any of the joints of the legs or arms. The movements were perfect and equal on the two sides of the body. The spine showed a postural scoliosis of mild degree, obviously the result of the inequality of the legs.

There was a slight inclination to hold the left shoulder higher than the right. The reflexes were normal. The heart was enlarged and a loud mitral murmur was to be heard. The urine contained a minute trace of albumin. Bence-Jones's albumose was not present. No focus of infection was discovered. A routine X-ray examination of the hips revealed the unusual character of the case, and led to an extensive radiographic investigation of the skeleton by Dr. Bertram Shires.

X-ray Findings.—The right half of the pelvis (*Fig.* 362), including the outermost part of the right half of the sacrum, and the right femur showed

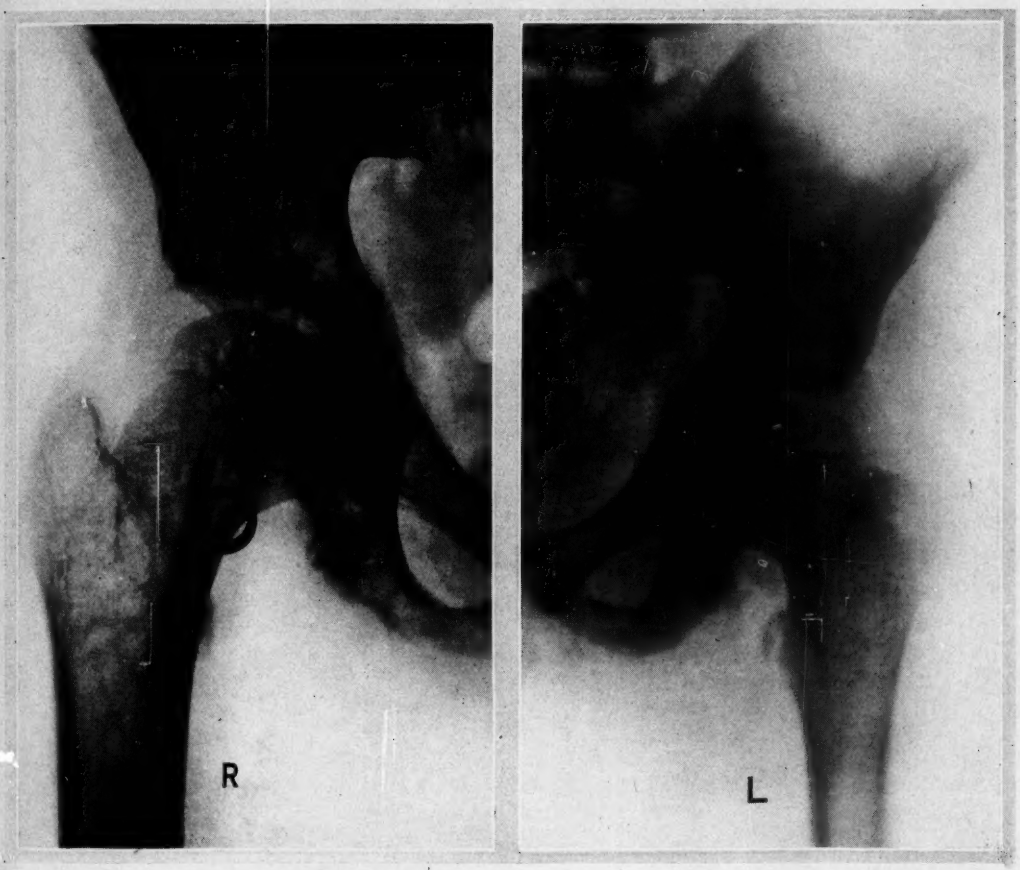


FIG. 362.—The bones about the hip-joint. (The bones on the two sides are really of equal size.)

a very marked difference from the corresponding parts on the other side. The general appearance of the bone seems to approximate more closely to what is usually termed ' atrophy ' than to any other abnormal condition. Here and there the appearances rather suggest osteitis fibrosa, but the similarity is only apparent at a few spots, and the gross changes in the bones as a whole are quite unlike this disease. Though atrophy seems to be the best word to describe the condition seen, the changes in the bone differ in several respects from those usually noted in atrophy associated with arthritis and disuse. There is a curious striated appearance, the lamellæ—

particularly those running up from the calcar femorale—standing out boldly against the bone around, which is less dense than normal. In addition, the femoral head and the bone adjacent to the acetabulum are mottled and sprinkled with dense spots. The shaft of the femur shows a solid tube of compact bone, not thinner than the normal as in an ordinary atrophic bone, but the interior of the shaft is less dense than that of the other side. The

FIG. 363.—The bones about the knee.

shaft of the right femur is at least as thick as that of the left. There is nothing to suggest that the joint surfaces and articular cartilages are affected: this agrees with the clinical findings. There is no alteration in outline of the bones. The right knee (*Fig.* 363) shows similar changes, most marked in the epiphyses of the femur and tibia. The longitudinal striation of the tibial shaft is worthy of note. In the ankles and feet (*Fig.* 364) the difference in density on the two sides is very striking. The striation is marked in all

the bones, while the dotted appearance is not very obvious, though certainly present in the epiphyses of the tibia and fibula and in the cuboid. The pencilling of the outlines of the bones is not so marked as in the typical atrophic foot. The right arm (*Fig.* 365) shows similar changes, but to a much less obvious degree. The upper end of the humerus shows the typical dotted appearance, which is in strong contrast to that of the left; the upper end of the shaft shows longitudinal striation. Changes are also seen in the acromion and glenoid region.

Sir Robert Jones and Dr. Thurstan Holland, who kindly took a great interest in the case, pointed out that the bones of the left arm and leg were

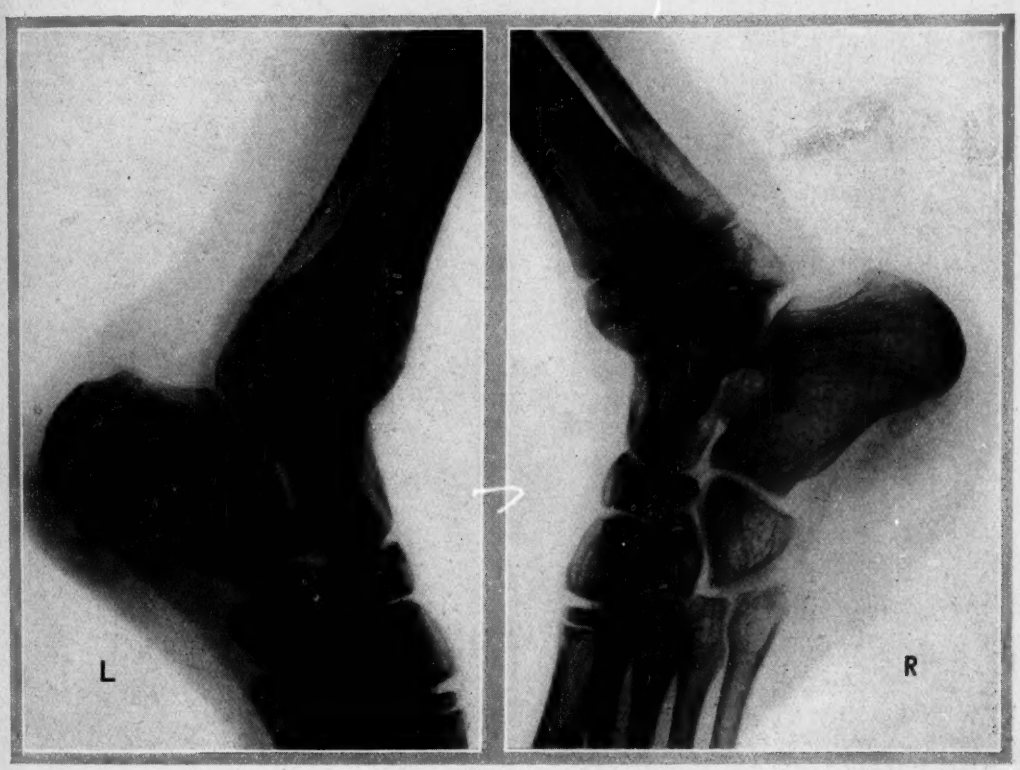


FIG. 364.—The bones about the ankle.

not quite normal, the changes being similar to those seen on the right side, but much less marked. There is not much to choose between the elbows; both ulnæ show striation at their upper ends. The hands show rather more distinct differences, the changes on the right being similar to those seen in the leg. The os magnum shows a large dense spot. The skull shows no changes except that in one antero-posterior view there is seen a density in the orbital regions, suggesting sclerosis or thickening of the great wing of the sphenoid on the right side. A second antero-posterior view failed to bring out this difference. Radiograms of the spine and ribs showed rarefaction changes in the head of the twelfth rib on the right side, but nothing abnormal otherwise.

To sum up the result of the X-ray examinations, we may say that the

right half of the pelvis, the right lower limb, and to a smaller degree the right arm, show an abnormal translucency to X rays. The chief lamellæ, however, stand out boldly, and in many places give an appearance of striation which is unusual. In addition a curious dotted appearance is seen in some epiphyses, and in the juxta-acetabular region of the os innominatum. Lastly, the bones of the limbs on the left side are not quite normal.

While it seems possible, or even probable, that the long period of rest incidental to the acute rheumatic attack might account for the abnormal

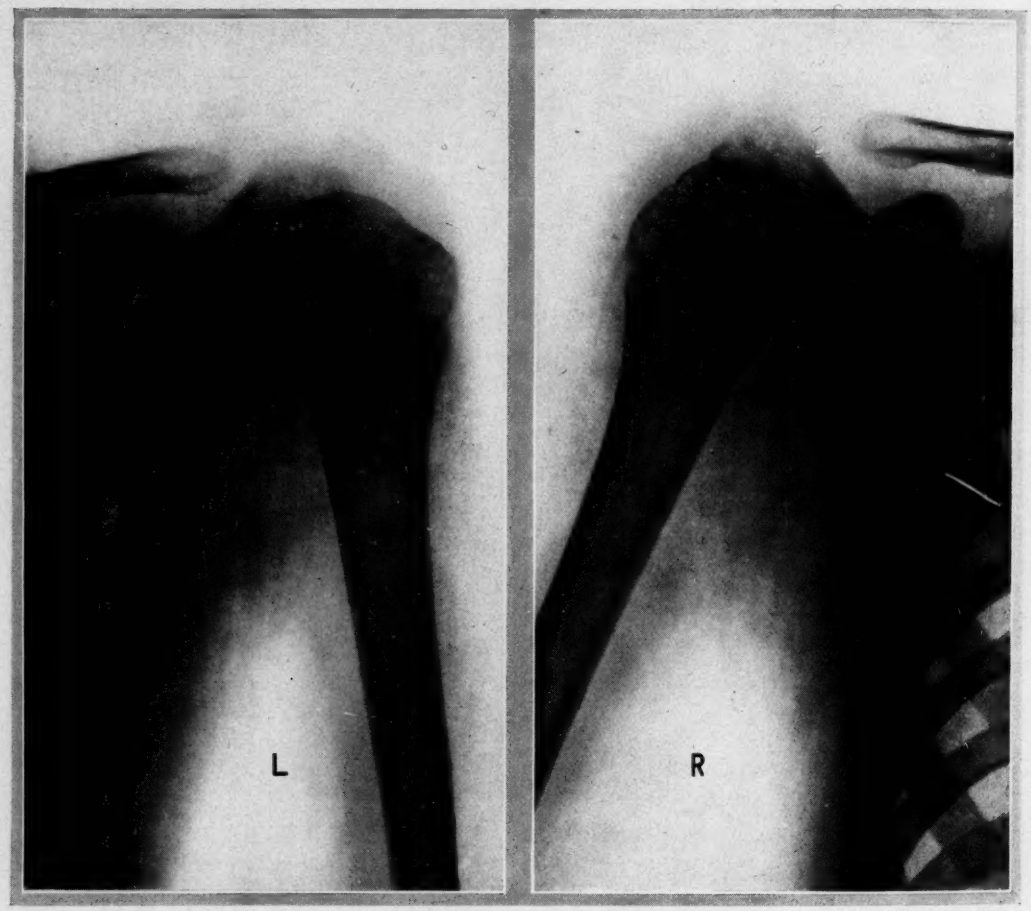


FIG. 365.—The bones about the shoulder.

atrophic changes seen in the left limbs, this would not explain the conditions seen on the right side. As already stated, fibrocystic disease of the bones is simulated only at a few places, the general appearance of the bones in this case being entirely unlike those seen in osteitis fibrosa. Compared with atrophy as usually seen, there are, as has been pointed out, several differences. Moreover, no cause for such atrophy of one side of the body has been discovered. Except for the very fleeting rheumatic infection, there is no history whatever of arthritis of any joint, and no history of rest or immobilization which should not have affected all four limbs to an equal extent. In this

connection it is interesting to note that both Sir Robert Jones and Dr. Calvé, in kindly writing to the author about the case, recall cases of arthritis of doubtful origin where the radiograms revealed changes of a somewhat similar kind, but these changes were localized to the region of the affected joint. So far we have not heard of a case where the changes were similar to those seen in this case and were equally widespread.

Dyschondroplasia has been suggested as an explanation. Dr. Thurstan Holland was kind enough to send us a reference to a paper on some extremely interesting experimental work on this disease carried out by Bentzon.[1] This is, it is true, an affection of the limbs of one side of the body, but the radiographic appearances are quite unlike those seen in this case. The masses of cartilage in the ends of the diaphysis of the affected bone are more or less columnar in shape, and, as Dr. Bentzon points out, these columns are arranged in a radiate manner from the point of entry of the nutrient artery towards the epiphyseal line. The radiate striation is quite unlike the longitudinal striation seen in this case. Moreover, in the two or three instances we have seen, the affected limb was shorter, not longer, than its fellow, and this would seem to be the general rule.

As regards the dotted appearance, we have a radiogram of a leg, from the case of a baby of one month, with a congenital short leg. In this leg all the epiphyses, as well as the tarsal bones that show any ossification, are stippled. These portions of the bony skeleton look as if a brush had been loaded with paint and flicked at them, so that instead of one centre of ossification there are a dozen or more. The diaphyses in this case were not obviously abnormal. Unfortunately the case was lost sight of. My colleague, Dr. Donald Paterson, once let me see a case where all the limbs showed stippled epiphyses; in this instance the diaphyses were abnormally short and stout, with enlarged ends. Except for the dotted appearance of the epiphyses, the bones in these two cases bear no resemblance to those in the case now being considered.

Osteogenesis imperfecta was also considered, but discarded as giving quite different radiographic appearances. The obvious strength of the tubular shafts of the long bones in this case is in marked contrast to the fragile appearance of the bones in the majority of cases of osteogenesis imperfecta. Moreover, this boy has never sustained a fracture.

Finally, we have to admit that we can offer no adequate explanation of the changes seen in this boy's bones. It would seem to be more than probable that the changes date from birth, or at any rate were present before the attack of acute rheumatism, and that they are not progressive. Time and further X-ray examinations can alone decide this latter point. Seeing that the affection is not a general one, no treatment, by drugs or otherwise, seems likely to be of the least use. As the affected leg is already longer than the other, local treatment to improve the nutrition of the limb seems to be contra-indicated.

As the heart condition demands abstention from all active exercise, and the bones have hitherto been equal to the strains put upon them, the boy has been allowed to return to school.

REFERENCE.

[1] Bentzon, P. G. K., "Roentgenological and Experimental Studies on the Pathogenesis of Dyschondroplasia (Ollier's Disease)", *Acta Radiologica*, 1924, iii, June 2, 89.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD.

THE CLINIC OF PROFESSOR CLAIRMONT IN ZURICH.

THE Clinic of Professor Clairmont is at the Kantonspital. It consists of about 230 surgical beds. Professor Clairmont has on his staff two 'first' assistants, and eight other 'official' whole-time assistants. In addition to this, at the

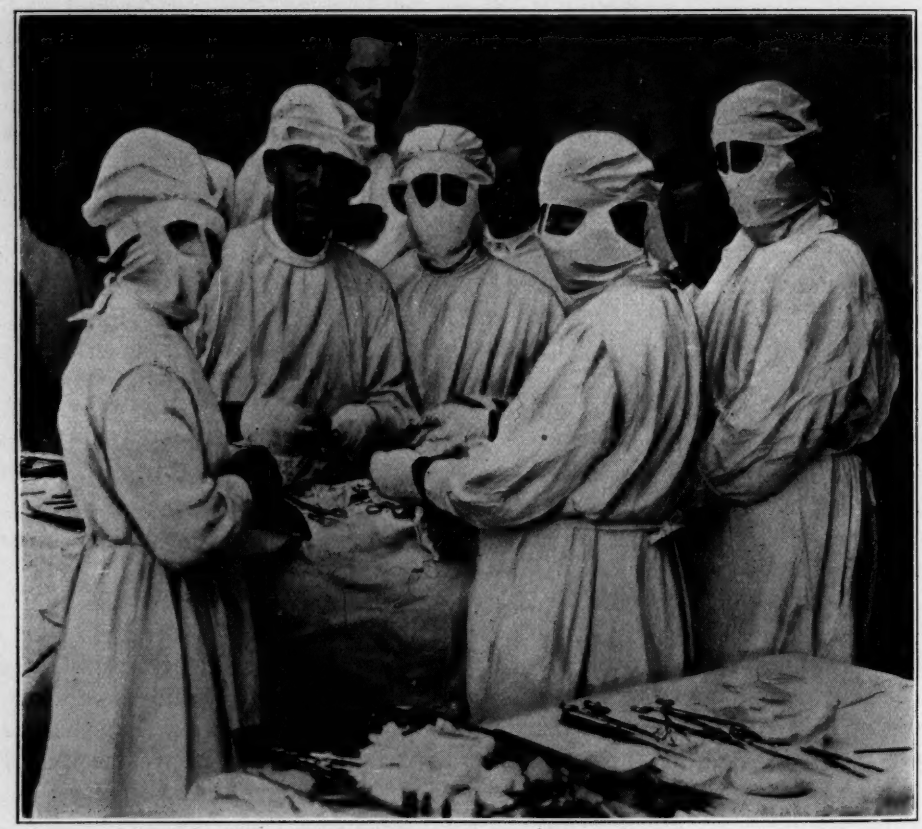


FIG. 366.—Professor Clairmont in his Clinic.

time of our visit he had five resident 'voluntary' assistants who, one presumes, are not paid, and also some non-resident 'voluntary' assistants.

The Clinic is the official one for the instruction of medical students. Every patient, whether in- or out-patient, pays something according to his

means. If too poor to pay, then his parish pays. Insurance companies have usually a contract with the hospital, and pay for the treatment of accident cases.

The programme of work at the Clinic during the two days spent there was extremely full and varied. Five operations were done each morning. Each afternoon was spent in visiting either the wards or subsidiary departments. The visit to the new Dermatological Institute was especially interesting.

The operations usually commence at 7.30 a.m. They were made more than usually interesting by each visitor being supplied the night before with a very detailed history of the cases, and also the results of the special examinations. Thoroughness was the key-note to everything seen at the Clinic.

We witnessed the following operations :—

1. **Second-stage Operation for Removal of Cerebellar Tumour, etc.**—(*Operation* 8.35 *to* 9.7.) The 'first stage' of the operation had been done on Sept. 5. The patient was a man, age 26, whose illness had commenced with giddiness and headache six months previously. The headache, at first diffuse, later had become localized in the occipital region. Vomiting had been frequent. Mental symptoms had been so marked that the diagnosis of dementia præcox had been made, and he had been sent into an asylum.

Soon after admission, marked cerebral symptoms had again appeared, and he was transferred to the surgical clinic. He complained of headache, and there was tenderness on percussion over the occipital region. He walked with an ataxic gait, and there was hypotonia of the muscles of the right leg. Both knee-jerks were increased, and Babinski's sign was present on the right side. The right eye could not be closed. Nystagmus was present, slow to the right and quicker to the left. There was paralysis of the right external rectus muscle. The corneal reflex on the right side was diminished, on the left side increased. The field of vision was contracted on both sides, but more so on the right. There was papillitis on each side, with retinal hæmorrhages. There were symptoms of irritation of the left labyrinth. The urine contained a small amount of albumin, and some red and white blood-corpuscles and *Bacillus coli communis* were present.

The diagnosis made was that of tumour of the cerebellum, probably in the upper part of the right vermis. At the first stage of the operation on Sept. 5, a large area of the occipital bone had been removed. The pericranium had been turned down with the skin-flap ; the bone had not been preserved. The dura mater was found to be bulging. At the end of the operation, which had been done under a local anæsthetic, the patient had lost consciousness. The pulse was then 160 per minute. Consciousness was quickly regained after the injection of caffeine and camphor.

The second stage of the operation, which we witnessed, was done under general anæsthesia by means of ether given by a Dräger apparatus ; regular readings of the blood-pressure and the pulse- and respiration-rates were taken by the anæsthetist and charted. The dura mater was exposed and found to be bulging. It was opened by a semicircular incision, and a cyst on the *left* side of the cerebellum was evacuated. The anæsthetist reported that he felt every manipulation of the brain by the operator affect the rate of the pulse. If pressure were made on the brain, the pulse was 80 per minute;

when pressure was removed, it was 120 per minute. A small portion of the cyst wall was removed for examination, and in a few minutes the pathologist reported that it was a gliosarcoma.

The dura mater was closed with separate catgut sutures, and the skin incision with separate silk sutures. The operation area was protected by a moulded papier-mâché splint, which was kept in position with a starch bandage. It was proposed to nurse the patient lying on his chest for the first twenty-four hours.

2. **Removal of the Right Internal Semilunar Cartilage.**—(*Operation* 9.20 *to* 10.10.) The patient was a male, age 24, who gave a typical history of injury with subsequent locking of the joint and synovial effusion. The operation was done under general anæsthesia. The operator stood on the left side of the patient; the leg was extended; no tourniquet was used.

An internal J incision was employed, the lower curve being across the tubercle of the tibia. It commenced about 2½ in. above the patella, and was about 6 in. in length. The skin was reflected and the quadriceps divided longitudinally in the line of the incision where the muscle-fibres join the tendon, thus exposing the capsule of the joint. The capsule was incised just anterior to the internal lateral ligament. The synovial membrane was then opened along the length of the wound. The knee was bent to a right angle, and an extensive view of the interior of the joint obtained. An adhesion between the retropatellar pad of fat and the anterior end of the internal condyle of the femur was exposed and divided. The internal cartilage had a 'bucket-handle' fracture, and was completely removed. The incision was closed in layers, fine silk being used throughout. After the dressings had been applied, the limb was put in a plaster-of-Paris gutter-splint which had been made previously, extending about 8 in. above and below the knee-joint.

In the after-treatment the limb is immobilized for seven to ten days on the splint. The splint is then removed and the limb rested on a cushion, active movement being allowed and massage given. Passive movement is not allowed until the end of the second week at the earliest. The average stay in hospital is five weeks, and work is usually commenced in eight weeks.

3. **Thyroidectomy.**—(*Operation* 10.15 *to* 11.15.) The patient was a woman, age 31. At the age of 15 she had had rheumatic fever and endocarditis. Since that time, in addition to the heart affection, she had had stiffness of the neck and had been unable to open her mouth fully. The goitre had been present since the age of 11. It had increased in size during each of her five pregnancies, and after confinement had gone down. The goitre had increased in size during the last two months, and was causing dyspnœa. She was not now pregnant.

Physical Examination.—The heart shows no dilatation, but there is a systolic murmur at the apex. The thyroid gland is diffusely enlarged, but more on the left side than the right. It is somewhat fixed to the deeper structures and moves only slightly on swallowing. There is dullness on percussion over the upper part of the sternum. A skiagram shows that the left auricle of the heart is enlarged; the trachea is shortened, and pushed over to the right, in addition to being slightly compressed. The cervical vertebræ show lordosis due to bony ankylosis between the articular processes of

the 2nd, 3rd, and 4th cervical vertebræ. There are rudimentary cervical ribs. The Wassermann reaction is negative. Laryngoscopy shows the vocal cords moving normally. The trachea cannot be seen.

Operation.—The anæsthesia employed was regional anæsthesia with novocain 1 per cent containing 5 min. of adrenalin in each ounce. The incision was the lower collar incision. The pretracheal muscles were divided transversely. The thyroid was exposed; the left inferior thyroid vessels were exposed as they entered the capsule. The vein was ligated before the artery. This step is considered to be important if any symptoms suggesting Graves' disease are present. The site of ligature was just outside the capsule.

The gland was then dislocated forwards, the capsule opened, and the vessels were ligated within the capsule as they entered the gland. The left superior thyroid vessels were ligated last. The left lobe was enucleated, the whole of the isthmus removed, and two-thirds of the right lobe resected. The raw surface of the remaining external third of the right lobe was cut in concave fashion, so that it could easily be covered by approximating the edges. The patient sat up to have the bandages applied.

So far as regards the regional anæsthesia employed, the experience of the Clinic was that in 50 per cent of the cases it was good, and 10 per cent were total failures.

4. **Thoracoplasty for Tuberculosis of the Right Lung with Cavity Formation and Hæmoptysis.**—(*Operation* 11.15 *to* 12.40.) The patient, a male, age 26, had pleurisy at the age of 11 months, and during childhood measles and scarlet fever. In April, 1922, he had been admitted to a sanatorium for progressive tuberculosis of the upper lobe of the right lung. On May 18, 1923, artificial pneumothorax had been induced on the right side on account of hæmoptysis, and he returned to work in April, 1924. In August, 1924, he had had four attacks of hæmoptysis. He also had pyrexia and night sweats. The sputum contained numerous tubercle bacilli. He had had a leucocytosis of 16,400. X rays show tuberculosis of the upper lobe of the right lung with cavity formation.

The operation was done under local anæsthesia with 1 per cent novocain. A long vertical incision was made parallel with, and about 1½ in. away from, the spinous processes. The incision commenced at the level of the 6th dorsal spine and ended at the 12th. A small portion of the posterior part of each rib, about 2 in. long, was removed, commencing with the 7th and extending down to the 10th. The portion removed was divided first anteriorly and then posteriorly. No attempt was made to include the angle. The skin incision was then continued upwards, and a similar portion of each of the remaining ribs removed, up to and including the 1st rib. The removal of the portion of the 1st rib was very difficult, and it had to be nibbled away with bone-cutting forceps.

A long rubber drainage tube with many lateral openings was laid along the whole length of the wound and brought out at the lower angle of the incision, the split muscles being stitched over it. The skin incision was closed, and the right side of the chest enclosed in an elastic bandage. The bandage was made of strips of elastic webbing, 2 in. broad, stitched at each end to adhesive strapping, which, being fixed to the skin, held it in position.

5. **Removal of Bullet from the Body of the 5th Cervical Vertebra.**—(*Operation* 12.50 *to* 1.50.) The patient, a boy of about 12 years, was shot accidentally by one of his playmates on July 16, 1924. The missile entered just below the angle of the lower jaw on the right side. When admitted to hospital there was profuse bleeding, and for five days he was unable to move his head in any direction. He had pyrexia; there was no paralysis, and no loss of sensation. He returned home on the sixteenth day after the accident. Since then he has had periodic attacks of severe pain in the neck radiating towards both shoulders.

Physical Examination.—The turning of the head is limited especially to the right side. Any attempt to bend the head backwards gives rise to severe pain. He can bend the head forwards quite normally. Pressure over the transverse processes of the cervical vertebræ is painful, especially on the right side. On palpation of the pharynx through the mouth, a small hard protuberance can be felt just above the larynx and to the right of the middle line. The protuberance, the size of a pea, is thought to be the bullet projecting from the body of the vertebra. The radiograph shows the projectile in the body of the 5th cervical vertebra, and also a fracture of the body of the 4th cervical vertebra.

Operation.—This was begun with a local anæsthesia, but later a general anæsthetic was administered. An incision about 3 in. long was made behind the right sternomastoid muscle, and the dissection carried down to the deeper structures. A scar was found between the pharynx and the prevertebral muscles. An unsuccessful search with the finger in the wound was made. The assistant could feel what was thought to be the projection of the bullet with the finger in the mouth, but it could not be reached by the operator, although bone was chiselled away. After searching unsuccessfully for an hour the operation was abandoned.

In the afternoon a visit was made to the Roentgen Institute of the Kantonspital. The Director, Dr. Schinz, showed a series of extremely good skiagrams of typical conditions, those illustrating gall-stones and those of the human fœtus being especially good. Dr. Schinz explained the difficulty in obtaining radiological evidence of the presence of gall-stones, even if calcium is present in the stone, by the fact that the wall of the diseased gall-bladder is often more impervious to X rays than the gall-stones themselves.

The department contains a very complete deep X-ray therapy plant, which was explained by Dr. Schinz and his assistants.

On Tuesday, Sept. 16, the second day of the visit, the following operations were witnessed:—

1. **Cholecystectomy.**—(*Operation* 7.45 *to* 8.40.) The patient, a woman, age 33, had been taken ill on Aug. 13, 1924. She was admitted on Aug. 17, with typical cholecystitis. The condition had not fully cleared up. It was a first attack. Under general anæsthesia the abdomen was opened by a vertical transrectal incision. The gall-bladder was distended and full of stones. The ducts were exposed after separating many adhesions with a blunt dissector. No stone was present in the ducts. The gall-bladder

was dissected off the liver, beginning at the fundus, and burst while it was being manipulated. The cystic artery was exposed, and tied with silk. The junction of the cystic and common duct was seen.

The gall-bladder was removed. The common duct was then fixed with two catgut ligatures to act as retractors, and the duct opened between the ligatures. Metal bougies were passed down the duct into the duodenum to dilate the sphincter muscle. The common duct was then closed with catgut, and the abdomen closed after a tube had been put down to the site of incision in the common duct.

2. **Suspected Duodenal Ulcer.**—(*Operation* 8.45 *to* 9.30.) The patient was a man, age 26, a decorator. In 1921 he had lead poisoning, with severe colic and vomiting after meals. He was treated at home, but he has never felt well since. He had a distended feeling after meals, and made himself sick in order to get relief. He continued working until the spring of 1924. From that time he has had vomiting, frequently after every meal, and constant pain in the epigastrium. The pain bears no relation to food; it is, however, worse when he is hungry. He has lost 22 lb. in weight since March, 1924.

Physical Examination.—He is a well-nourished man with diffuse goitre. There is tenderness in the epigastrium 1½ in. above the umbilicus, but no rigidity. There is no lead line on the gums. The gastric analysis shows increased acidity. Occult blood was present in the stools only once out of four examinations. The X-ray report states that there is a superficial duodenal ulcer.

Operation.—Under general anæsthesia the abdomen was opened in the middle line. No ulcer on the duodenum or stomach could be found. No gall-stones were present. The appendix showed chronic inflammatory changes and was removed, the stump being cauterized with a Paquelin's cautery. The abdomen was closed in layers with discontinuous sutures.

3. **Cancer of the Stomach.**—(*Operation* 9.40 *to* 11.) The patient was a tailor, age 64. Eighteen months ago he had discomfort in the stomach, which was relieved by food. He was treated medically for three weeks and felt better. He continued work until six weeks ago, when he again had dyspepsia and loss of appetite. He has no nausea or vomiting. He has a sensation of fullness one or two hours after meals; he has a disgust for meat. He thinks he has lost weight during the past three weeks.

Physical Examination.—The abdomen is retracted. The liver margin can be felt 2 in. below the right costal margin. There is a large tumour palpable on the right side of the abdomen, which does not move on respiration but can be differentiated from the liver. Gastric analysis shows free acid to be present, and a total acidity of 42; no bile, no lactic acid, and no blood is present. Occult blood is present in the stools. X rays show a small residue after four hours. The shape of the stomach is like a hook.

Operation.—Under general anæsthesia the abdomen was opened in the middle line. There was a large cancer of the pyloric end of the stomach, which owing to the fixity was deemed to be inoperable. The liver had no metastatic growths present. The operation performed was a 'pyloric exclusion'. The stomach was divided into a proximal and distal portion, the latter including as much of the lesser curvature and the glands as possible. The distal

portion was then closed and the proximal portion attached to the jejunum, which was brought up through the transverse mesocolon, as in a Polya operation.

Professor Clairmont considers 'pyloric exclusion' a better operation than a palliative gastrojejunostomy. Vomiting is delayed for a longer period. No complete statistics could be given as to the prolongation of life, etc., in this operation as compared with the prolongation after gastrojejunostomy.

A case of obturator dislocation of the hip-joint having been admitted, this was then reduced under a general anæsthetic. There was no difficulty in the reduction. The skiagram of the dislocated hip was excellent.

4. **Enlargement of the Prostate.**—(*Operation* 11.30 *to* 12.15.) The patient, age 68, was quite well up to six weeks ago, and had never had difficulty with micturition. He first had retention after drinking a quantity of cold water, and had to be admitted to the Inselnspital at Berne. He was catheterized for a fortnight while in hospital, and then a catheter was tied in, the bladder being washed out twice each day. After a week he was transferred to the Kantonspital at Zurich on Aug. 25, 1924.

On admission the bladder was distended and contained 700 c.c. of turbid urine. The urine contained pus and blood, and was infected with *Bacillus coli communis.*

Cystoscopic Examination.—This shows a shallow post-prostatic pouch. The urethral orifices are normal, but between them is a small tumour the size of a cherry (adenoma of subtrigonal glands). The mucosa of the bladder is swollen, blood-vessels are not clear, and some trabeculation is present. The kidney function as indicated by test is normal. Blood nitrogen is normal. Wassermann reaction is negative.

The pre-operative treatment has been carried out by tying in a catheter and giving urotropine by the mouth. The anæsthesia was by tropococaine (1½ c.c. of 5 per cent solution) by the spinal route.

Operation.—The bladder was opened suprapubically and the contents were siphoned off. After trying for half an hour to shell out the prostate with the finger, some 'bands' had to be divided with scissors. The tumour then shelled out easily. A two-way tube was put into the bladder and the latter stitched tightly round it.

The after-treatment consists of continuous irrigation of the bladder for twenty-four hours with iced potassium permanganate solution. No catheter is passed at any time unless there is difficulty in micturition.

5. **Tuberculous Osteitis of Symphysis Pubis, with Sinus Formation and Mixed Infection with the *Staphylococcus aureus.***—(*Operation* 12.30 *to* 1.10.) The patient was a woman, age 28, who had been admitted on Sept. 3, 1924. In 1919 she had had tuberculous glands of the left side of the neck, and treatment with X rays had given very good results. Four weeks before admission she had complained of pain and swelling in the pubic region, more especially on the right side. She continued her work, but at the end of August she consulted a doctor, who tapped the swelling and removed a glassful of brown pus. During the last few days the swelling has increased in size and become painful. A discharge of pus has occurred at the site of the puncture wound. There has recently been pyrexia and a feeling

of illness, but no pain or frequency of micturition. The bowels have been regular.

Physical Examination.—The skin over the lower part of the abdomen is tender and œdematous. There is a rounded, reddened swelling over the mons veneris, from the middle of which pus is oozing from a sinus. The genitalia are normal. The urine contains leucocytes but no micro-organisms. No tubercle bacilli have been found even after animal inoculation. The Wassermann reaction is negative. Culture of the pus from the sinus shows *Staphylococcus albus.* X rays show a tuberculous osteitis of the symphysis pubis with sequestrum formation.

Operation.—Operation was directed towards the radical excision of the whole of the infected area. This involved a wide excision of the soft parts and the right half of the symphysis pubis.

The procedure was criticized by those present as being unnecessarily drastic, and it was suggested that sequestrectomy alone would have been sufficient. Professor Clairmont disagreed, and stated that in his experience only the widest possible excision of the disease was sufficient. He admitted that it would be a long time before the patient could walk, and probably a special support to the pelvis would always have to be worn.

During the morning a demonstration of the method of blood transfusion employed in the Clinic was given by Dr. Ochsner. Dr. Ochsner, who was from Chicago, had been working in Professor Clairmont's Clinic for a year, while one of Professor Clairmont's assistants had been exchanged for him and was working in Professor Ochsner's Clinic in Chicago.

The method employed was that of Percy as used in Professor Ochsner's Clinic in Chicago. It consists of giving whole blood by means of Kimpton's tube. All patients have their blood grouped before operation. The donors are obtained from patients in the wards suffering from fractures or similar disabilities. These cases are all grouped. There is a list of suitable donors put up in the theatre each day; consequently, if an emergency transfusion is required, it can be done at once. There is no difficulty in getting donors. After the transfusion the donors are put on extra diet with wine, etc. No fee is paid to them in money.

In the afternoon cases were demonstrated in the wards, and then a tour of the new Dermatological Institute of Zurich was made under the guidance of the Director, Professor Bruno Bloch.

This Institute has just been built and equipped at a cost of £100,000. Before the money was spent, the Government took a referendum of the people as to whether they thought it justifiable to spend so much money on such an Institute. Professor Bloch himself went round the towns and villages lecturing. He demonstrated to all classes the necessity of having in their midst a thoroughly up-to-date Dermatological Institute and Clinic. He illustrated his lectures with the beautiful wax models he has in the Institute. These faithfully portray the most important and common skin diseases, particularly those due to syphilis. The referendum gave a large majority in favour of spending this amount of money.

The Clinic contains 110 beds, and is probably one of the most completely

equipped of its kind in the world. There is a special department for the treatment of scabies, any case of which Professor Bloch stated he could cure in two hours. The police bring up most of these cases. The laboratories are very complete, consisting of histological, bacteriological, and chemical departments. There is a special fungus department, and of course a radiological department; the whole Institute possesses 130 mgrm. of radium element.

Many experiments on animals are performed, and some of the results of exposure of rabbits to X rays, and mice to tar inunction, were shown. The rabbits develop epithelioma of the ear after exposure twice a week for about two years. The epithelioma is fatal by producing general metastases in the lung and elsewhere. The mice are affected with cancer in two or three months "if the tar is good". The method adopted is to paint the abdominal wall with tar solution twice a week.

Research work appeared to be progressing actively in all departments of the Institute. All the work witnessed at Zurich can only be regarded as most thorough and painstaking. No effort is spared in arriving at a correct diagnosis. There is a striking spirit of camaraderie and enthusiasm between the Professor and his assistants which can be felt to permeate the whole of the Clinic.

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF GALL-STONES WITH A GUMMA OF THE LIVER MISTAKEN FOR MALIGNANT DISEASE OF THE GALL-BLADDER.

By CECIL P. G. WAKELEY, London.

The following case is of interest because there was nothing in the family or clinical history to suggest a syphilitic lesion of the liver. The growth in the liver was very hard and was adherent to the gall-bladder, which contained numerous stones; a diagnosis of malignant gall-bladder seemed justified, as no primary lesion could be discovered in the abdomen. Had the gall-bladder and the growth not been removed, but the abdomen closed without further surgical interference, the case would have been labelled malignant and a gloomy prognosis given. The patient would no doubt have recovered, and might easily have stated that the cancer had been cured by some faith-healing device or by some special cancer cure.

It is in such ways that the cancer-cure statistics may be very fallacious. This case also shows the importance of a routine Wassermann reaction being performed, for had a positive reaction been returned before instead of after the operation, excision of the gumma would probably not have been performed.

The history of the case was as follows: Mrs. M. A. T., age 43, came up to the out-patient department of King's College Hospital in March, 1924, complaining of pain referred to the right side of the abdomen which radiated through to the back. The pain commenced in September, 1923, and had gradually become worse, so that the patient was kept awake at night. The pain was more acute at some times than others, and on two occasions the patient had vomited. The pain had no relation to food, although she thought that her trouble was indigestion, and it was treated as such by her doctor.

The patient was married and had two children, both of whom were healthy girls. No history of miscarriage. Her periods were regular and normal. On examination, the abdomen moved well on respiration; the liver was found to be slightly enlarged, being about an inch and a half below the costal margin. A hard, tense swelling could be palpated just below the ninth costal margin; it was tender to the touch. No other abnormality could be palpated in the abdomen. The case was diagnosed as one of gall-stones, and operation was advised. On May 30, a laparotomy was performed under gas and oxygen anæsthesia; a supra-umbilical incision was made just to the right of the middle line, the rectus muscle was exposed and drawn outwards. On opening the

peritoneal cavity, the gall-bladder was exposed; it was found to be enlarged, thickened, and contained several stones. Just to the right of the gall-bladder a hard lump projected from the under surface of the liver, which appeared to be attached to the gall-bladder. There was no perihepatitis in the region of the tumour, and a diagnosis of carcinoma of the gall-bladder was made. The stomach, intestines, and pancreas showed no sign of abnormality; in fact, the abdomen seemed to be quite normal with the exception of the gall-bladder and liver.

It was decided to remove the gall-bladder together with the tumour in the liver. The lower end of the cystic duct was divided between clamps and the cystic artery secured. The lump in the liver was completely excised by means of a diathermy cautery knife. There was very little bleeding. After excision of the tumour in the liver, the raw area was packed lightly with white gauze and the abdomen closed. The gauze plugging was removed after two days, a small piece of corrugated rubber drainage tubing being inserted instead. This was removed on the sixth day as there was no discharge. The wound healed quickly and convalescence was uneventful, the patient losing all her pain.

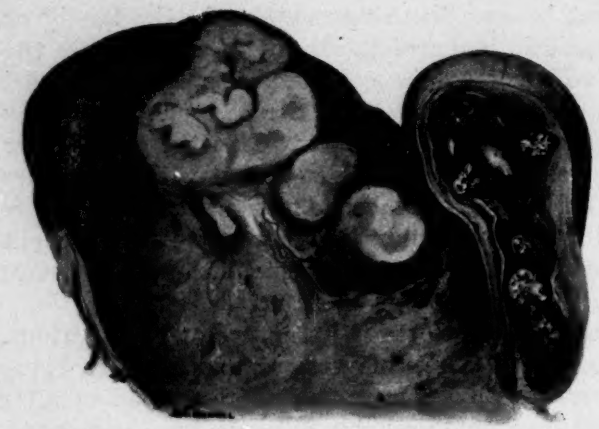


FIG. 367.—Specimen showing excised portion of liver with the gall-bladder.

On section of the excised portion of the liver, one large and two smaller hard white areas were seen (*Fig.* 367). The larger one approached up to the margin of the liver. The gall-bladder was very thickened and contained numerous stones. Several sections were taken from different parts of the excised portion of the liver for microscopical examination; the pathologist was asked to report if malignant disease was present. The report was as follows:—

> The sections present the typical appearance of a gumma of the liver. The sections from the mass of the gumma showed areas of coagulation necrosis, surrounded by syphilitic granulation tissue in which were a few giant-cells. The sections from the surrounding liver tissue showed a marked perivascular infiltration with lymphocytes.

After receiving the pathologist's report, a Wassermann reaction was performed and was reported as strongly positive. Antisyphilitic treatment was commenced before the patient left hospital and has been continued since. The patient has completely lost all her pain, and has put on half a stone in weight since leaving hospital.

LOOSE BODIES IN THE ANKLE-JOINT.

By GEORGE H. STEVENSON, Glasgow.

The patient, a man, age 22, was admitted to my wards at Bellahouston Hospital early in May, 1924.

History.—While on military service in India during November, 1920, he fell when running down a hill and twisted his right ankle. The injury was not considered serious enough at that time to necessitate his admission to hospital and he was treated by his regimental medical officer. The joint has remained painful from that time onwards, and he states that the initial swelling had never completely disappeared. Recently a 'lump' on the inner

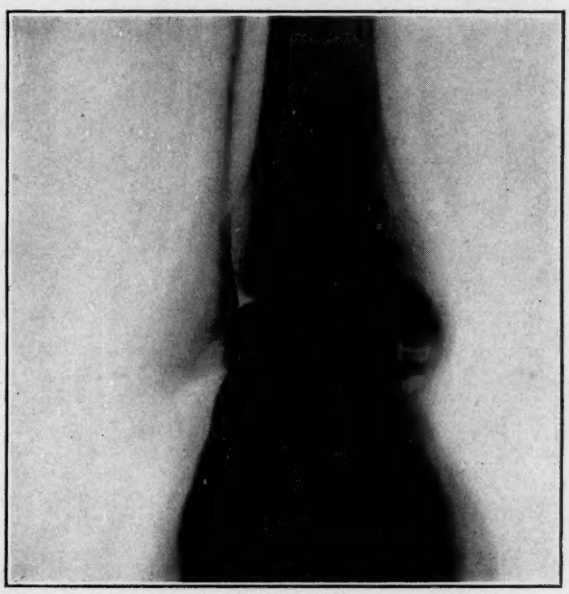


Fig. 368.—Antero-posterior view of ankle-joint, showing size and position of loose body.

side of the joint had been troubling him, and this had apparently been growing larger.

Examination.—There was a hard swelling below and behind the internal malleolus of the right ankle. The swelling appeared to extend upwards under the malleolus, and was painless on pressure. All movements of the joint were quite free and there was no sensation of grating. All the other joints appeared quite normal. X-ray examination showed a large shadow (*Figs.* 368 and 369) in the region previously indicated, having the characteristic 'ghostlike' appearance associated with loose bodies in the knee and elsewhere.

Operation.—A J-shaped incision was made over the swelling posterior to the internal malleolus, and the tendons of the tibialis posticus and

flexor longus digitorum, together with the posterior tibial nerve and artery, were retracted backwards after division of the internal lateral ligament. The capsule of the joint was then opened, and a large loose body was easily extracted, followed by a second of much smaller size. The loose bodies had apparently gradually distended the small sac of synovial membrane found in this region.

On removal, the larger of the two bodies measured $1 \times \frac{3}{4}$ in., the approximate size and structure being well illustrated by the X-ray photograph (*Fig.* 370). The smaller body was slightly larger than a pea.

FIG. 369.—Lateral view of ankle-joint, showing loose body.

FIG. 370.—Radiograph showing approximate size and texture of the larger loose body.

I cannot find any reference to loose bodies in the ankle-joint in the literature at my disposal, and none of my colleagues can remember having seen a similar case. Loose bodies occur commonly in other joints, but it seems remarkable that a body of the size described should have been able to form in a joint having the comparatively strict limitations of the ankle, and it is for this reason I put the case on record.

I am indebted to Dr. Henderson and Mr. Livesy for the excellent X-ray photographs, and to the Ministry of Pensions for permission to publish the case.

STRANGULATED HERNIA THROUGH THE FORAMEN OF WINSLOW.

By W. G. McKENZIE and DUNCAN WOOD, Bristol.

The following case is of interest from its rarity and for comparison with other reported cases.

The case was that of a man, age 59, previously healthy, who had not suffered from indigestion. He first complained of discomfort, which rapidly passed to definite and constant pain, immediately after the midday meal. The bowels had been open that morning. There was no vomiting. He was first seen two hours later.

On examination, the abdomen moved freely; there was little or no tenderness on palpation, but it was slightly tympanitic to percussion; pulse and temperature were normal.

The following morning, i.e., seventeen hours after onset, pain was more severe, and localized to the epigastrium; there was definite abdominal distention, tenderness on deep palpation in the epigastrium, no rigidity; pulse and temperature were normal. No vomiting, no passage of fæces or flatus to turpentine enemata.

Twenty-four hours after onset the pulse was 120, the tongue clean, and there was no vomiting; the abdomen was distended in the neighbourhood of the umbilicus; the upper third of the abdomen was rigid and gave the sensation of a swelling being present; this swelling was resonant to percussion; the normal liver dullness was present.

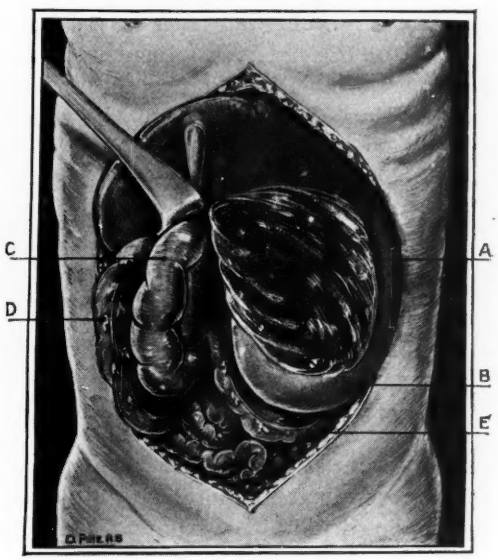


Fig. 371.—Hernia into foramen of Winslow. A, Gangrenous loops of jejunum in lesser sac; B, Stomach pushed downwards; C, Dilated loop of small intestine entering foramen of Winslow; D, Collapsed ascending colon; E, Transverse colon pushed downwards.

Operation was performed three hours later. On opening the peritoneal cavity, blood-stained fluid escaped. The stomach and some loops of small intestine were found distended. On drawing the stomach downwards, a large black swelling was seen to be pushing forwards the gastro-hepatic omentum. The latter was thin, and easily split, showing strangulated loops of small intestine lying in the lesser sac. On tracing back the strangulated loop, it was found to be entering the lesser sac through the foramen of Winslow (*Fig.* 371). This foramen was dilated further with the finger, and the strangulated loops partly pushed and partly drawn through the foramen out of the lesser sac. The released gut did not revive, thrombosis having

already taken place. Forty inches of small intestine were resected and an end-to-end anastomosis was done. Owing to the serious condition of the patient, no attempt was made to close the foramen of Winslow or the gastro-hepatic omentum.

Abdominal distention persisted after the operation in spite of pituitary injections and repeated turpentine enemata. Death occurred from paralytic ileus on the third day after operation and the fifth day of the disease.

The following points are of interest :—

1. Onset after a midday meal in a previously healthy individual is similar to the cases of Elliot[1] and of Treves.[2] The rate of onset appears to vary considerably. In Haw's[3] case the history was only half an hour, death being apparently due to shock from the large amount of bowel strangulated. In Rawlence's[4] case operation was not called for before the sixteenth day.

2. The absence of vomiting before operation in a small-gut obstruction was a marked feature. In Treves's case vomiting did not start until the second day, and was then only slight in amount. In Rawlence's case vomiting commenced on the thirteenth day. This may possibly be explained by the distended coils round the foramen of Winslow pressing on the pylorus and preventing regurgitation of the duodenal contents into the stomach.

3. Operation. Authorities differ as to the best method of reducing the strangulated loop. Moynihan[5] recommends opening the lesser sac through the gastro-hepatic or gastrocolic omentum, and then reducing the strangulated loop ; or, if this is found to be difficult, to do a temporary enterotomy first, after which, having emptied the contents of the loop, reduction will be possible.

If the damaged gut is too friable for manipulation, a portion of adjacent healthy gut could be drawn into the lesser sac through the foramen, and the loop resected *in situ*. Jeanbrau and Riche[6] advise enlarging the foramen of Winslow by incising the peritoneum forming the anterior lamina of the gastro-hepatic omentum as in exposing the common bile-duct. This, whilst easy in the post-mortem room, would have been extremely difficult in the present case on account of the pressure of the dilated small intestine obscuring the foramen. Treves, in the account of his case, states that the presence of distended coils of intestine in the vicinity of the opening rendered the demonstration of the relations very difficult.

In the authors' case the strangulated loop when released could be drawn well outside the abdominal cavity for excision. The resected loop was jejunum ; no abnormality of the colon was noticed.

REFERENCES.

[1] Elliot Square, *Brit. Med. Jour.*, 1886, i.
[2] Treves, *Lancet*, 1888, Oct.
[3] Haw, *Lancet*, 1909, i, 1598.
[4] Rawlence, *Jour. R.A.M.C.*, 1918, xxx, 332.
[5] Moynihan, *Retroperitoneal Hernia*, 1906.
[6] Jeanbrau and Riche, *Rev. de Chir.*, 1906, No. 4.

A CASE OF MYOMA OF THE STOMACH WITH GASTRODUODENAL INTUSSUSCEPTION.

By L. E. BARNETT, Dunedin.

Myoma of the stomach is regarded as the most common non-malignant tumour occurring in this situation. Many of the cases recorded were complicated by some degree of intussusception, and the following case is an example of an extreme degree of that remarkable condition.

W. S., age 54, a powerfully-built farmer and stock dealer, was referred to me by Dr. P. Gow, of Milton, Otago, in February, 1924, with the provisional diagnosis of carcinoma of the stomach. He had been suffering for two years from gastric pains, vomiting, and occasional hæmorrhage, with loss of weight and increasing weakness. A firm, rounded mass the size of a goose's egg was sometimes palpable, sometimes not, in the right hypochondrium. Gastric analysis indicated the presence of much mucus and blood and slight diminution in hydrochloric acid.

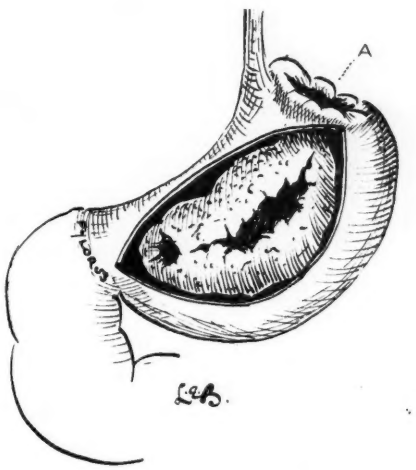


Fig. 372.—Diagrammatic sketch showing tumour as seen when stomach was opened. A, Intussusception beginning.

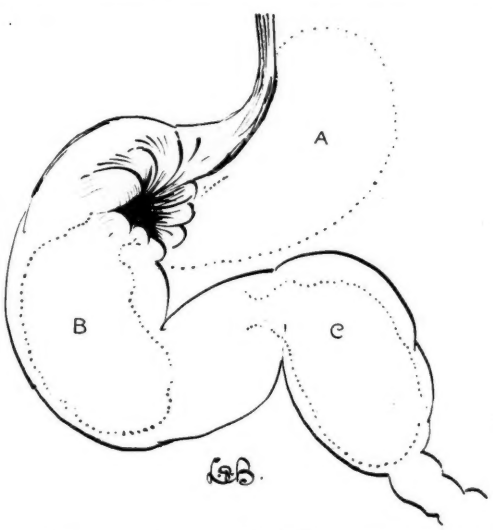


Fig. 373.—A, Stomach intussuscepting; B, Tumour as felt in duodenum; C, Tumour as felt in jejunum.

X-ray examination showed only dilatation and some stasis.

Operation on Feb. 20, 1924, revealed the presence of a large oval mass inside the hugely distended duodenum. By manipulation this mass could be pushed onwards through the whole length of the duodenum, and for a distance of 20 cm. into the jejunum. It could also be easily manipulated back into the stomach, and on investigation it was found to be a tumour attached by a pedicle 2 cm. in diameter to the cardiac end of the stomach, in close proximity to the œsophageal opening. Its extraordinary freedom of movement was permitted by an unusual degree of intussusception of the fundus of the stomach. The illustrations (*Figs.* 372 to 376) give a better idea of the condition than any written description could do.

The stomach was freely opened and the tumour cut away from its attachment without difficulty. The part of the stomach wall involved in the process of intussusception was of a peculiar leathery consistence, due, no doubt, to chronic fibrosis. The tumour itself showed two deep crateriform ulcers. A similar appearance is figured in other cases of gastric myoma that have been published.

FIG. 374.—Photograph of tumour after removal.

The patient made an uneventful recovery and rapidly regained his former weight and robust appearance.

The report of Dr. P. P. Lynch, Clinical Pathologist to the University of Otago, is as follows :—

" Tumour was roughly ovoid—12 cm. by 6 cm. by 5 cm. At its upper and posterior part there was a pedicle 2 cm. in diameter. Its anterior aspect was excavated, showing an irregular cavity with narrow elongated opening and ragged, overhanging edges. The surface of the tumour was covered by epithelium derived from stomach. The floor of the excavated portion was lined by granulations only.

FIG. 375.—Myoma, low power (× 11).

FIG. 376.—Structure of myoma, high power (× 150).

" Microscopic examination of the tumour showed masses of large, smooth muscle cells with well-supported vessels, and in parts many points of

hæmorrhage. The tumour was definitely capsulated, this capsule being formed for the most part by thickened submucosa. It is a myoma with central necrosis and excavation."

An excellent account of benign tumour of the stomach was published in the Mayo Clinic *Transactions* for 1921.

I am indebted to Dr. Lynch and Mr. McKay, of the Pathological Laboratory of the University of Otago, for the sections and photographs.

ADENOMA OF SMALL INTESTINE WITH INTUSSUSCEPTION.

By G. M. KENDALL and I. W. CORKEY, Epsom.

The rarity of the condition and the difficulties of diagnosis make the following case worthy of note.

A married woman, age 36, was first seen on April 5, 1924. She complained of attacks of pain with vomiting; she had had a similar but milder illness about a year before. The pain was referred to the upper abdomen and passed round to the back; it was at times relieved by vomiting. She sometimes woke up in the early morning with the pain; there was no distinct connection with the taking of food, but it had on occasion been brought on by a cup of tea. At this time physical examination was completely negative. The bowels were not constipated. These attacks of pain continued at intervals, but vomiting ceased.

About three weeks later the character of the pain changed and was now felt in the lower dorsal part of the back, and passed round towards the epigastrium. There was no vomiting. The attacks of pain were very severe; the bowels were constipated. At this time, on some days a distended viscus was felt passing across the abdomen just below the umbilicus; this was thought to be the transverse colon, but on May 10 slow peristaltic waves were seen passing from left to right and beginning to the left of and about the level of the umbilicus. The passage of these waves coincided with a spasm of pain, and pressure on the viscus caused similar pain. In the afternoon of May 18 a more severe attack began and persisted; the patient vomited several times during the night; a tumour now became palpable below the umbilicus.

The abdomen was opened on May 19. A recent intussusception was found, three feet in length, situated in the lower part of the ileum but not reaching the ileocæcal valve; it was easily reduced. At its apex was a pedunculated tumour of the size of a small tangerine orange; the glands in the mesentery near were moderately enlarged. The piece of gut containing the tumour, and part of the mesentery containing the glands, were excised, and an end-to-end anastomosis was done. The surface of the tumour was necrosed and inflamed.

Dr. J. F. Taylor examined the tumour microscopically, and reported:—

"The tumour is an adenoma, with œdema, congestion, hæmorrhage, and inflammation, evidently from torsion of the pedicle. The glands show chronic inflammation only."

The patient has made a good recovery.

REVIEWS AND NOTICES OF BOOKS.

Lectures on Gonorrhœa in Women and Children. By J. JOHNSTON ABRAHAM, C.B.E., D.S.O., M.A., M.D. (Dub.), F.R.C.S. (Eng.), Surgeon, London Lock Hospital; Senior Surgeon, Kensington General Hospital, etc. Crown 8vo. Pp. 142, with 9 illustrations and 4 plates. 1924. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

THIS book supplies a long-felt want, and should be of considerable value to those interested in this particular line of work. In the foreword the author states that his object in publishing the lectures is to interest the general practitioner and to bring to his notice methods of examination and treatment of the disease in its early stages (when it is curable) without the use of any special instruments or skill beyond that possessed by the average practitioner. This, of course, is most commendable, and is an object greatly to be desired, for the majority of women prefer to consult their doctor rather than visit a specialist or attend a venereal clinic. We feel, however, after reading the book, that it will be long before Mr. Abraham's ideals are realized, for there are few practitioners, particularly with panel practices, who can spare the time to learn and carry out the technique so ably set forth. It is unfortunately a notorious fact that gonorrhœa in women is a neglected disease—neglected by the patient, the practitioner, and the gynæcologist; therefore if this book stimulates only a few to practise the methods of examination and treatment advocated, the benefit will be reaped by many. In explaining the methods of examination, the author rightly insists on the dorsal decubitus and on the importance of staining smears taken from the labia, the urethra, the cervical canal, etc.; he also points out the important fact, of which many are unaware, that for legal purposes staining by Gram's method is essential.

The first chapter contains a clear and concise description of the symptoms and treatment of the disease in its acute form, including an excellent account of treatment of vulvitis in children which should be of great value to house officers and others. Stress is laid on the importance of realizing that acute endocervicitis is the commonest symptom of gonorrhœa in the adult female, and that consequently the usual vaginal douche is useless. Of the three varieties of treatment described, the author considers that McDonagh's manganese-contramine method yields the best results.

Chapters 2 and 3 deal with the complications of the urinary and genital tracts. In the former the modern methods of treating cystitis and pyelitis are described, and in the latter the technique and value of ionization and diathermy for chronic endocervicitis are clearly explained.

Gonorrhœal toxæmia, septicæmia, and the important extragenital complications such as synovitis, arthritis, and so on, are described in two chapters on metastatic gonorrhœa. In discussing the treatment, the author, in our opinion very rightly, warns the practitioner against topical treatment of the cervix or uterus during the acute stage of the complication, and advises that it should be deferred for several weeks after the acute symptoms have subsided. "Concentrate on the joints while they are inflamed, and attend to the primary focal lesion afterwards" is sound advice.

The chapters on gonococcal ophthalmia and gonococcal sterility are excellent, and the latter in particular contains much valuable information.

The final chapter, on tests for cure, is perhaps one of the most important in the book, and sets forth very clearly the tests which the author has found to be the most reliable from practical experience.

The book contains several useful illustrations, is clearly written, and amongst its many merits possesses one which should appeal greatly to those for whom it is intended—it is brief and to the point.

Internal Derangements of the Knee-joint: their Pathology and Treatment by Modern Methods. By A. G. TIMBRELL FISHER, M.C., F.R.C.S., late Hunterian Professor, R.C.S. of England, 1921–2; Assistant, Surgical Unit, University College Hospital. Demy 8vo. Pp. 144 + xii, with 80 illustrations. 1924. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.

MR. TIMBRELL FISHER'S book is probably the most complete account of internal derangements of the knee that has been published in English. It is particularly strong on the pathological and anatomical side, and the possible or probable mechanics of injuries of the semilunar cartilage are considered in very great detail.

The author makes much point of the difference in strength of attachment of different portions of the internal semilunar cartilage and their bearing upon injuries. There has been much divergence of opinion upon the importance of the internal lateral ligament in this connection, and discussion whether rupture of this ligament is a necessary concomitant of a lesion of the internal semilunar cartilage. Mr. Timbrell Fisher holds that it is not so, and that, in fact, an intact internal lateral ligament is essential to the production of certain lesions of the cartilage, because the latter is firmly attached to the posterior or short fibres of the ligament, a weak spot existing in the cartilage just in front of this attachment. The mechanism of production of injuries of the cartilage cannot yet be said to be cleared up, but Mr. Fisher has certainly added a new mechanical suggestion, and many clinicians will agree with him that rupture or over-stretching of the internal lateral ligament is not an essential part of a cartilage injury, even though they may not agree that injury commonly starts at the weak spot he has described.

If criticism is here made of certain other aspects of the book, it is rather with the suggestion that in some subsequent edition improvements may be introduced. The clinical side of the book is not quite up to the same standard as the pathological side. Hardly sufficient attention is given to the difficulties of differential diagnosis, more particularly in those cases seen by the surgeon between attacks when the knee is apparently perfectly normal. These cases continue to worry the surgeon, who is obliged to arrive at his diagnosis almost entirely upon the history that he can obtain from the patient. Another important point that might be elaborated is the attempt to make before operation a differential diagnosis of the exact lesion of the cartilage likely to be found. This is more particularly important in lesions of the posterior part of the cartilage, which are not visible through the ordinary anterior incision, and which, when they are suspected, necessitate either a wide anterior incision or the removal of a cartilage, apparently normal in front, in the expectation that the posterior half will be found fractured, or a second exploratory incision in the posterior part of the joint. The latter point—that is, the second incision over the posterior part of the cartilage—now not uncommonly adopted by surgeons, is omitted from the book altogether.

Mr. Fisher's patella-displacing operation is a very valuable one for complicated and difficult explorations of the knee-joint; it has undoubted advantages over the patella-splitting method and does less eventual damage to the knee-joint.

Keeping to the clinical side: after a displacement of the cartilage with locking of the knee which has been reduced by manipulation, rest for fourteen days with massage and graduated exercises is recommended. Theoretically this sounds a rational method, but at the same time Mr. Fisher, as the result of his pathological investigations, seems to have come to the conclusion that a split cartilage does not unite. If this is so, there appears to be no reason for maintaining this period of rest, and it seems agreed that in many cases a knee which has been completely locked, reduced by manipulation, and used forthwith, remains permanently free from any subsequent derangement. The question of what actually happens when a displaced cartilage is reduced is a very important one. Is the cartilage in most cases really restored to its normal position; or is it pushed into some other position in which it gives no trouble; or is it further fractured, so that the free ends no longer get into the way in movements of the joint? If rest is to be advocated in order to try to get healing, fourteen days seems to be too short a time. Six weeks or longer should be necessary on pathological grounds, yet recurrence of displacement undoubtedly occurs after quite long periods of splinting.

There is, of course, much included in the book besides injuries of the semilunar cartilage. Mr. Fisher's work on loose bodies is so well known already that it hardly requires mention. It is briefly recapitulated here in so far as it concerns the subject. The book is very profusely illustrated; the illustrations are exceedingly well reproduced and very appropriate to the subject-matter. It is a book which ought to be in the hands of every operating surgeon who ever opens a knee-joint, and it is to be hoped that in due course, as the result of further experience and consideration, Mr. Fisher will produce a second edition which will give still greater clinical assistance.

Minor Surgery and Bandaging. By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Crown 8vo. Pp. 423 + viii, with 239 illustrations. 1924. London: J. & A. Churchill. 10s. 6d. net.

THIS is the eighteenth edition of Heath's *Minor Surgery and Bandaging*. Whilst intended for the use of house surgeons, dressers, and junior practitioners, it is such a storehouse of practical 'tips' and suggestions that it will prove of great value to the experienced surgeon. It provides an excellent and safe bridge over the great gap between the time of qualification—a time of, perhaps, great theoretical knowledge, but very little wisdom in actually dealing with patients—and the time of having become an experienced house surgeon. It serves, too, as an invaluable guide for students on first working in the surgical department of their hospital.

The book has been ably revised and brought up to date, and there is no modern technique in general use without mention or description. The chapters on fractures are particularly good. In spite of the introduction of much new matter, the careful pruning of obsolete methods, together with the use of thin paper, render the volume of convenient size. The general arrangement and illustrations are excellent, and the index is particularly good and clearly arranged.

The Science and Art of Anæsthesia. By Colonel WILLIAM WEBSTER, D.S.O., M.D., C.M., Professor of Anesthesiology, University of Manitoba Medical School. Royal 8vo. Pp. 214, with 37 illustrations. London: Henry Kimpton. 24s. net.

THIS book aims at being a concise manual for the occasional anæsthetist, and, although a short work, it is, on the whole, fairly comprehensive. As would be expected, ether and nitrous oxide and their combinations are the most completely described, with the latest methods and machines for their use. Chloroform is very briefly discussed, although, in this country at least, it is probable that it is still the routine anæsthetic in use for the smaller operations in general practice. The physiology of anæsthesia is very well described, and sound advice is given in the chapters on the selection of the anæsthetic and on the nature of the operation. Surgical shock and the various necessities before and after operation are also well but briefly described.

It is an interesting and readable book, and very well got up, but the price, twenty-four shillings, appears a little large for what the author admits should be supplemented by larger text-books.

Chirurgie de l'Oreille, du Nez, et du Pharynx. By Dr. GEORGES LAURENS. 2nd edition, entirely revised. Royal 8vo. Pp. 1052, with 733 illustrations. 1924. Paris: Masson et Cie. Fr. 100.

IT may be said at once that this work of 1052 pages is the best which has appeared on the subjects with which it deals. This eulogium holds good whether it is applied to the excellent illustrations, of which there are 792, to the paper and type, and above all to the wisdom and fine judgement which the author has brought to the fulfilment of his task.

It will be a most helpful volume to all teachers of oto-rhino-laryngology, and of priceless value to senior clinical assistants and those about to confine themselves to these branches of surgery. These will do well to read again and again the "General Considerations", which occupy fifty-seven pages and deal with such important

subjects as the best type of installations, sterilization of the field of operation, instruments, and hands, and general and local anæsthesia; the latter is greatly favoured in France, and consequently its technique has been highly perfected. We are not acquainted with a better account than the author presents of the principles of radiotherapy (including X rays), and of the attempt to classify the histological structure of tumours according to their radio-sensibility. The reasons given for choosing radium or X rays for different circumstances are obviously the result of prolonged experience.

With regard to all the operations described an excellent plan is followed. Each procedure is described with its indications, technique, and results. The contra-indications for intervention are clearly stated.

Under surgery of the ear the statement (p. 65) that the operations of aural surgery, major and minor, may be performed under local anæsthesia, although a general anæsthetic is *justifiable* in resection of the mastoid for tumours, for labyrinthine disease, and cerebral abscess, will scarcely receive assent in this country, where the art of general anæsthesia is so highly developed. On the other hand, a conscientious student of Dr. Laurens' work will certainly gain the impression that English-speaking nations would do well to exploit to a much greater degree the possibilities of local or regional anæsthesia as induced by a mixture of novocain and adrenalin.

The various accidents and difficulties met with in the mastoid operation are well described and illustrated, and valuable hints given as to avoiding or overcoming them. Among these may be mentioned the wounding of the facial nerve, the lateral sinus, and the external semicircular canal. The author warns against deliberate denudation in radical operations on the mastoid unless there are definite indications for doing so. He thinks that non-observance of this rule may be the explanation of certain cases of chronic headache which follow the operation. In operations where the lateral sinus is infected, he states that statistics show an equal number of cures whether the jugular be tied or the sinus alone dealt with.

The operative treatment of labyrinthritis and of facial paralysis, the cerebral complications of otogenic origin, and tumours of the ponto-cerebellar angle are models of clear exposition and wise judgement.

Rhinologists will agree with the stress laid by the author on meddlesome interference after endonasal operations instead of allowing the wounds rest and natural progress towards cure (p. 383). In the treatment of intractable epistaxis we are surprised that no mention is made of the value of submucous resection of the deviated septum as a method for relieving the stretched mucosa in which the thin-walled vessel is often found to be situated. The surgical treatment of 'ozæna' is recommended, and consists in narrowing the nasal fossæ by forcing inwards the inner walls of the maxillary sinuses. Ramadier's procedure is described in detail. The description and illustrations of the surgical treatment of the diseases of the nasal sinuses could scarcely be improved upon. Local or regional anæsthesia is advocated, and the hints on after-treatment should be read by every rhinologist.

Space will not permit a review of the author's views with regard to general anæsthesia for the removal of adenoids. Whenever any general anæsthetic is administered, "the fatal dose may approach the useful dose, the precipice skirts the road . . . " He considers tonsillotomy a good operation in the general run of cases, and insists that there is no need to have recourse to a "veritable glandular castration", especially in young subjects. Considerable space is devoted to hæmorrhage following tonsil enucleation. It would have been well if the distinguished author had emphasized the necessity of ligaturing the tonsillar branch of the posterior palatine artery in every case. Post-operative bleeding would then be a very rare occurrence. He rightly describes galvano-cauterization of the tonsil as likely to "enclose the wolf in the sheepfold".

The chapters on the larynx and trachea, including endoscopy of these regions and the œsophagus, reach the same high standard as those which precede them. The major operations for malignant disease of the larynx and lower pharynx are beautifully illustrated, and the advantages of carrying them out under local anæsthesia are strongly urged and almost refute adverse criticism.

Collected Papers of the Mayo Clinic. Edited by Mrs. M. H. MELLISH. Vol. XIV, 1922. Royal 8vo. Pp. 1394, with illustrations. 1923. London and Philadelphia: W. B. Saunders Co. Ltd. 63s. net.

THE most weighty of the papers in this volume are those of which W. R. Meeker seems to be the inspiration, alone or with others, on "Paravertebral Nerve-block Anæsthesia". These enter fully into the indications for, and the technique of, this procedure in the different regions of the spine; but the indications for, and advantages of, trans-sacral nerve-block anæsthesia as compared with novocain spinal analgesia would be helpful. As the author of one of these papers admits, trans-sacral nerve-blocking usually requires a general anæsthetic and involves a delay of fifteen minutes. This comparison will not apply, of course, in the surgery of the chest or neck, where most of us do not follow Jonnesco in his advocacy of spinal analgesia.

Adson contributes two papers on "Removal of Tumours of the Spinal Cord" and "The Radical Treatment of Trifacial Neuralgia", both beautifully illustrated. Does the author underestimate the difficulties of approach to the Gasserian ganglion owing to variations of the middle meningeal artery? If he has met with such difficulties, it would surely be helpful to advise his readers how to deal with them.

J. C. Masson deals with "Post-operative Ventral Hernia" on sound lines. In his description of the technique for closure of such defects we think he does not sufficiently emphasize the importance of free excision of all scar tissue and approximation of healthy fibrous layers—for we do not attach any importance to muscle approximation, to which he allots a separate heading as an important point. We regret that he should make reference to the use of fascial transplants as advocated by Gallie and Le Mesurier, which for the purpose under discussion is surely unnecessary and clumsy.

Two papers by C. H. Mayo and Judd show the value of ileostomy for the removal of acute obstruction and as a safety-valve for resection of the colon, though in the latter instance we are not convinced that it is preferable to cæcostomy.

Every surgeon interested in the thyroid problem must at frequent intervals take steps to maintain his surgical sanity by reference to papers on the pathology of excess and diminution of this gland's activity, for there is scarcely any field in which it is more easy to do bad work unless in every case indications for surgery are based on a sound pathology. We feel, therefore, that the brief extract of Dr. L. B. Wilson's paper on "Hyperthyroidism" is well timed and well worth digestion, as are all his papers on this question.

H. I. Lillie's warning that earache is not necessarily of aural origin is one that cannot be repeated too often, and the same author's paper on "Infection of the Lateral Sinus" is valuable because he is able to include nineteen cases of this rare complication of mastoid disease. We are glad to find that he holds a very open mind on the value of ligature of the jugular vein.

Collected Papers of the Mayo Clinic. Edited by Mrs. M. H. MELLISH. Vol. XV, 1923. Royal 8vo. Pp. 1377, with 410 illustrations. 1924. London and Philadelphia: W. B. Saunders Co. Ltd. 63s. net.

THE Editorial Committee of the Mayo Clinic are to be congratulated on having the courage to change their policy with regard to the publication of the collected papers of the Clinic. Originally it had been intended that the volumes should contain reprints of all papers issued during the year, but the material has so much increased that this was found impossible. In the volume under review many articles without clinical interest appear only by title, and many more in the briefest possible abstract. It may be argued that it is extremely difficult to select for exclusion papers on researches which indirectly may prove to be of great clinical importance, and some of the abstracts are so terse as to be almost indigestible; but the net result of the change is satisfactory, for the volume is more comprehensive and is much more easily handled than those published in the last few years.

If no fact of great significance has yet emerged from the numerous papers on experimental work in connection with the formation of gastric and duodenal ulcers, yet many of the conclusions or hypotheses which have resulted from these studies are in the highest degree suggestive. We are therefore glad to see a further paper by Professor Mann, which shows that diversion of the secretions which neutralize the gastric acid, either by duodenectomy or transplantation of the ducts, results in the formation of typical ulceration.

The reprint of Dr. C. H. Mayo's paper on gastroduodenostomy will be that for which most surgeons will consult this volume, and it is to be hoped that the method suggested indicates a return to more rational and less mutilating surgery for limited and innocent lesions of the stomach and duodenum.

Dr. New reports 107 cases of actinomycosis, but it is unfortunate that the paper loses its general clinical value by being confined to observations of the disease as it affects the head and neck. It has long been recognized that superficial or peripheral actinomycotic lesions yielded to various treatments; but when the abdomen or chest is involved, the prognosis in our experience is very grave, and it would have been valuable to learn from the general experience of the Mayo Clinic whether their statistics show that there is any improvement in this class of case.

Etude du Syndrome douloureux du Côlon proximal. By Dr. OSCAR MERCIER, of the Faculté de Médecine de Paris. Royal 8vo. Pp. 248. Illustrated. 1924. Paris: Amédée Legrand.

THE literary form of a medical thesis is a convention of almost universal acceptance; the contention to be maintained, its past history and development, its anatomical, physiological, and pathological aspects, its relationship to other authorities friendly and inimical, and the final addendum of clinical material, more or less second-hand, as the foundation upon which the superstructure has been reared—these are its constant ingredients. In France apparently there is the added grace of a dedication—in this instance to twelve people specifically named, in addition to "mes maîtres de la Faculté de Médecine de l'Université de Montréal".

"Dans ce travail nous limitons notre étude à la constipation résultant d'un état morbide du côlon droit" is the opening sentence of this work—an admirable *motif* that becomes obscured later on amidst most of the ills that flesh is heir to. The reader is not spared "les troubles du système neuro-végétatif", "les déséquilibrés du système chromaffin", "vagotonisme", and "les déséquilibrés des glandes endocrines". Little more need be written to indicate the general tenor of the work. A deplorable confusion between the cæcum and the right colon pervades the book from beginning to end; in fact, very little at all is said about the colon, and "which is which one can never make out"! An operation always alluded to as 'cæco-transversostomie', recommended and designed by Professor Desmarest, whose cases form the basis of the thesis, is illustrated by a sketch in which 'colo-colostomie' is being performed obviously. This was done upon 9 patients out of the total series of 55, with the result that in 6 of them "la constipation persiste entrécarpée de débâcle diarrhéique". Upon 16 others Friedrich's old-fashioned operation of hemi-colectomy was performed, of whom 1 died, 3 were unrelieved, and 1 was too recent to be classified as a result. The remaining 30 cases were treated solely by the removal of the appendix and the resection of an anomalous group of tie-bands. In 11 of these no improvement followed the operation, in 4 there was slight improvement, whilst in the remaining 15 cases in which success was achieved it is stated that gross disease of the appendix obvious to the unaided eye and confirmed by microscopical investigation was present.

The "syndrome douloureux du côlon proximal" is not recognized easily amongst these cases. The views and experiences of Grégoire, Duval, Roux, and Wilms of the value of fixation of the proximal colon are lightly brushed aside—a mental attitude of the inexperienced that presents a not unfamiliar aspect—whilst Mercier's experience of his own methods summarized above seems to afford but exiguous evidence upon which to base any conclusion about anything. No doubt a grateful

Alma Mater will have added already this work to her 'archives des *Thèses*', whence within but a short time the dust of the poppy of oblivion will be but lightly stirred by the plumed brush of the library attendant. And, after all, why not? "Où sont les *thèses* d'antan?"

The Theory and Practice of the Steinach Operation: with a Report of 100 Cases. By Dr. Peter Schmidt (Berlin). Translated by Dr. M. D. Eder, and with an Introduction to the English Edition by J. Johnston Abraham, C.B.E., D.S.O., M.A., M.D. (Dub.), F.R.C.S. (Eng.). Crown 8vo. Pp. 150 + xiv. 1924. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

Professor Eugen Steinach is a biologist and laboratory worker of Vienna. He has been reflecting and experimenting for several decades. Steinach's work has nothing in common with that of Voronoff. His original results obtained by vaso-ligation in old rats are sufficiently striking. After the vasa had been tied in decrepit old rats, the animals took on a new lease of life; the fur grew again, they put on fat, and became agile and pugnacious. Obviously in some way the factor producing the male hormone had become augmented. Steinach discovered that after the operation the sperm-forming cells atrophy, but that the interstitial cells proliferate.

The book contains a review of the history and literature of the subject, but most readers will probably be interested chiefly in the details of the clinical cases, which are carefully recorded and are certainly remarkable. For example, a man prematurely old and incapable of doing his ordinary work in life is enabled by this very simple operation of vaso-ligation to become again a useful member of the community. It is a mistake to press unduly the recovery of sexual potency, which alone would not appear to some to justify even a slight operation. Amongst Eastern peoples an opposite opinion would probably be expressed. The fact remains that in *carefully selected cases* the whole body receives an impetus to what has not been inaptly described as rejuvenation. The same results have occurred in man after operation as Steinach previously noted as happening in decrepit old rats. And surely this need not be a cause of wonder if we remember the marvellous functions of the other endocrine glands, such as the thyroid, the suprarenals, and the pancreas, the knowledge of which has been gradually unfolded to our mental vision since the time of Brown-Séquard.

Many will read this book and gain a new interest in the experimental or Hunterian method of research.

L' Ostéosynthèse dans le Mal de Pott. By James George Kopp. Royal 8vo. Pp. 184, with 12 illustrations. 1924. Amsterdam: N. V. Drukkerij.

This monograph on the treatment of Pott's disease of the spine consists of a general description of the disease, its anatomy, pathology, symptoms, and treatment; but in particular there is a detailed account of 58 cases in which operative treatment was employed with a view to bringing about spinal fusion. The majority of these cases were operated on by Professor Noordenbos. In the earlier cases Albee's method was used, but in the great majority Halstead's operation was performed. This consists in cutting the spinous processes away from the laminæ, leaving them attached to the muscles on one side, and placing a tibial graft between the laminæ and severed spinous processes. All the operations were done under local anæsthesia (½ per cent novocain). In 12 cases out of the 58 death followed at an interval varying between seven months and six years. In all cases it is claimed that a rapid recovery with sound fusion of the spine was the immediate result of the operation. No extravagant claims are made as to the influence of the graft on the cure of abscess or paraplegia.

Practically no attempt is made at mechanical fixation of the spine by any external apparatus. Possibly this is the explanation of the fact that there have been several cases of fracture of the graft.

The monograph affords a useful and fairly written account of a considerable series of cases treated by one surgeon.